

Annual AVMA Meeting

Denver, Colorado

August 14-18, 1960

Journal

OF THE
AMERICAN VETERINARY
MEDICAL ASSOCIATION



FIRST REPORT—apes naturally infected with chicken pox. Page 256



Vol. 136

March 15, 1960

No. 6

Introducing



*for rapid
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BREVANE is truly a short-acting barbiturate — combining high anesthetic activity with low toxicity. Induction is smooth, rapid, and free of excitement or irritability. Dosage is low, requiring only 25 mg. (1 cc. of a 2.5 per cent solution) per five pounds of body weight.

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Kittens and Mink Have Vaccine in Common

Veterinarians with fur grower clients are using Feline Distemper Vaccine to protect mink against the virus of mink enteritis.

Antigenically, the virus of mink enteritis is identical or closely related to the virus of feline panleucopenia. Therefore, it is not surprising that inactivated panleucopenia virus vaccine of feline tissue origin (Norden) effectively immunizes against the serious enteritis problem in mink. According to a Minnesota veterinarian who has a fur grower client, the results in mink are "very satisfactory."

Do you have any fur grower clients?

Foaming Uterine Bolet Eases Unpleasant Task

In cases of retained placenta, try Borofoam Bolets (Norden) at the beginning of the "cleaning" procedure. While deodorizing, they reduce the slickness of membranes and aid manual removal of placental shreds. Upon completion, administer additional Borofoam Bolets or Sulfa Urea Bolets (Norden) for continued antibacterial action. Dosage is one to three bolets for cows and one for sows.

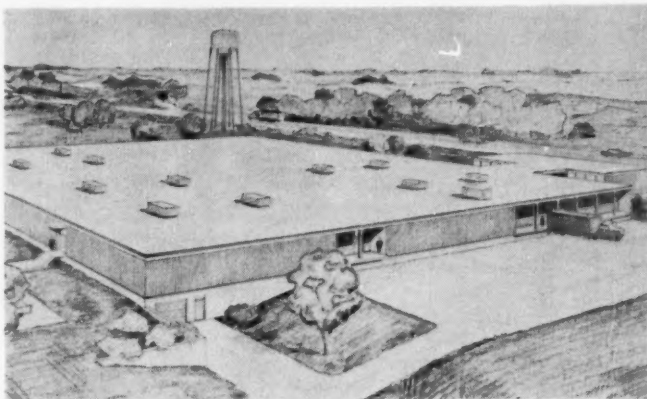
Milk Fever Therapy Pleases Dairyman

A North Dakota practitioner, called to treat a cow, found that the animal had calved 10 days earlier but had just gone down. The farmer, suspecting milk fever, had administered 1,000 cc of straight 25% calcium gluconate without response.

The veterinarian diagnosed parturient periesis and started treatment with Norcalciphos (Norden). Before 370 cc were administered, the cow got up, to the surprise of the farmer and gratification of the veterinarian. In this drought area with poor quality hay, the combination of Ca, P, and Mg and dextrose in Norcalciphos did the trick.

Especially indicated in complicated cases, Norcalciphos is also valuable in problem herds to help prevent relapses. And because the product is exceptionally stable there is no troublesome precipitation, even after freezing.

Norden Laboratories was issued the first license by the U. S. D. A. for the production of Cl. Chauvei-Septicum Bacterin (Vaccinol®).



Norden Starts Building Program

Construction of Modern Biological Production Unit to Begin Immediately

To accelerate its production program of veterinary biologicals and pharmaceuticals, Norden Laboratories, Inc., announces plans for

a new building program to start immediately. Bids for the first phase covering the biological production unit have been let.

Detail plans covering the remainder of the building program, including pharmaceutical production, warehousing and general offices, are being drawn.

When completed, the Norden plant will provide many new facilities dedicated to the production of biologicals and pharmaceuticals exclusively for the veterinary profession.

Site of the new building is a 138 acre plot two miles northwest of Lincoln, near the municipal airport, affording prompt service to all points of the United States. Currently on the site are research facilities, including buildings and holding pens.

Announcement of the building program was made simultaneously with the completion of the merger of Norden Laboratories, Inc., with Smith Kline & French Laboratories of Philadelphia.

Balance Important In Hog Cholera Vaccines

Extreme potency without safety would be disastrous in hog cholera vaccination—as would be extreme safety without potency. In NORVAC potency and safety are in balance for solid, durable immunity without interruption of steady gains.

There's a reason too for NORVAC's longer, stronger immunity. Tissues are selected only from pigs which have shown a definitely prescribed reaction following injection with modified virus. Each pig must have a normal white blood cell count and temperature prior to inoculation. After inoculation, only those animals are used which show a prescribed temperature curve and leukopenia pattern during the incubation period.

Iomycin Case Reports

Bovine pneumonia: Although treatment seemed hopeless, Iomycin was injected. Next morning the owner called to say cow was up and eating. A return call was not necessary. Texas.

Feline pneumonia: Prognosis in case of feline pneumonia was poor.

Yet recovery was complete following Iomycin therapy. California.

Gangrenous bovine mastitis: Cow with two gangrenous quarters was given two treatments consisting of Iomycin and Tri-Sulfa-G. Four days later the cow was well on the road to recovery. Ohio.

Journal

OF THE
AMERICAN VETERINARY
MEDICAL ASSOCIATION

Vol. 136 No. 6 March 15, 1960

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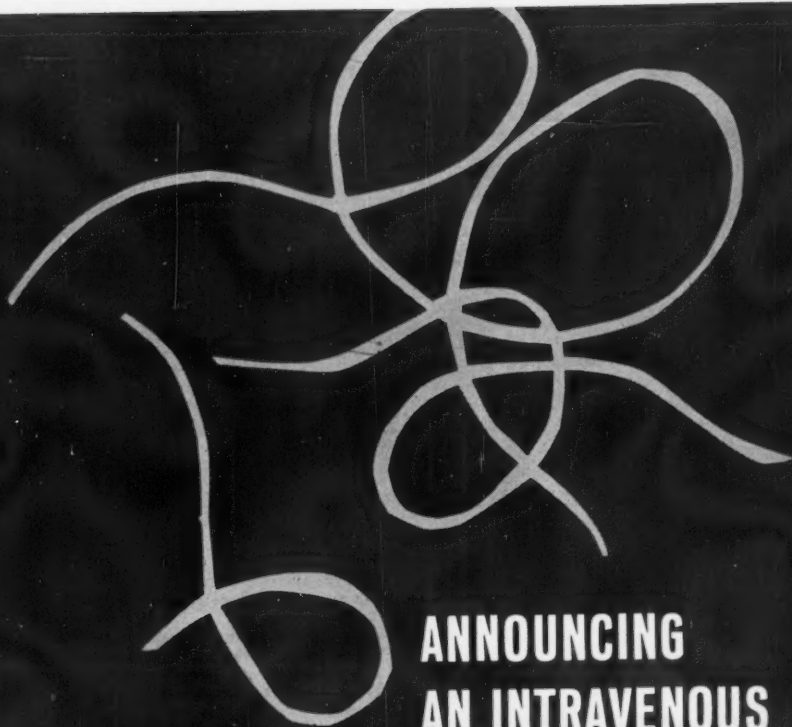
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... FULL ANTHELMINTIC EFFECT—NOTHING LOST THROUGH VOMITION
WHIPCIDE...the first intravenous anthelmintic...has proved highly effective against canine whipworms (*Trichuris vulpis*)...affords the following advantages:¹ / intravenous doses of 250 to 500 mg./kg. produced clearances in the 97 to 100 per cent range / intravenous Whipcide cannot be lost by vomiting / food present in the GI tract does not interfere with drug's action / dogs offer little or no resistance to IV administration ■ A recent study² shows that ova of *T. vulpis* usually disappeared completely by the third post-treatment day.

Now supplied 100 mg. multiple-dose vials.

1. Eshenauer, R. W.; Smith, S. R., and Ehrenford, F. A.; J.A.V.M.A. (Dec. 15) 1977; 2. Ehrenford, F. A., Nepler, R., and Johnson, R. Can. Vet. Med. 54:105-4-10 (Oct.) 1980



PITMAN-MOORE COMPANY

DIVISION OF ALLIED LABORATORIES, INC., INDIANAPOLIS 6, INDIANA

Correspondence

Feb. 9, 1960

Dear Sir:

Referring to the item "Antibiotics for Malignant Edema" (JOURNAL, Feb. 1, 1960: 128), I would like to report that I, too, have successfully treated malignant edema in a horse. This horse was kicked by another horse, the calks of the shoes having made a wound by tearing and puncturing. The infection became apparent about 2 weeks later.

Initially, I gave 6 million units of procaine penicillin, 5 Gm. of dihydrostreptomycin, and 10 cc. of Bicillin. Except for the latter, treatment was repeated on four successive days.

s/C. P. DILL, D.V.M.
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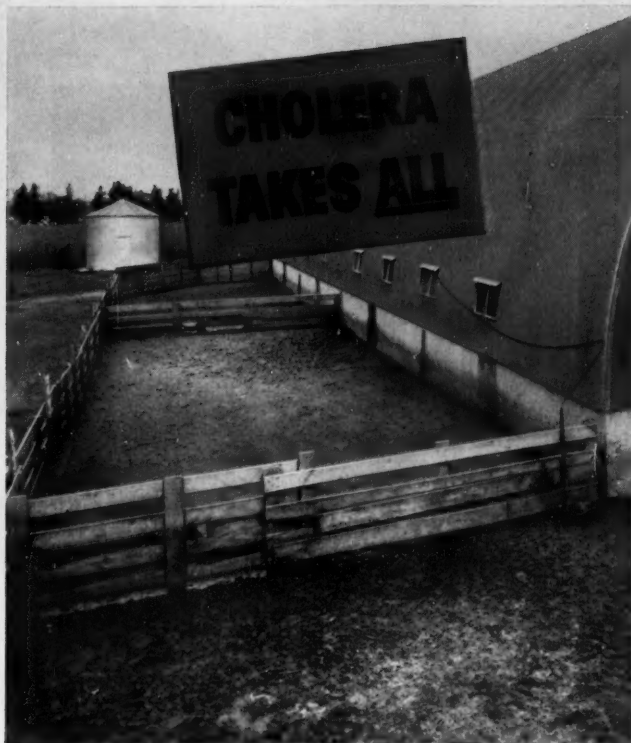
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You risk losing not just low-priced pigs, but all the corn, care & cash you've put into them!

Cholera so often strikes when pig losses are most costly

If you could settle for losing only a few small pigs—or sacrificing only your rather slim pork profit—maybe you could take a gamble on cholera.

But it's not just pigs—and it's not just profits—that hog cholera robs you of. It's the corn, care and cash you've put into those pigs. It's the high investment in feed, time and housing. That's what makes cholera costly.

Cholera is a threat the year 'round. It can strike in any month. But cholera hits peaks in late summer and early fall—and again in the spring. Its greatest risks come when your investment in swine is at its peak. The "ALL" that cholera takes is too much to risk. By comparison, vaccination of pigs is cheap. It is your only real protection.



Protect your stake
—VACCINATE!

Cholera struck on many hog farms in 1959. And again in 1958. Often in swine or areas having had no cholera in 10 years or more. That it did not strike more times in more places is to the credit of vaccination—in large measure to professional vaccination with M.L.V.

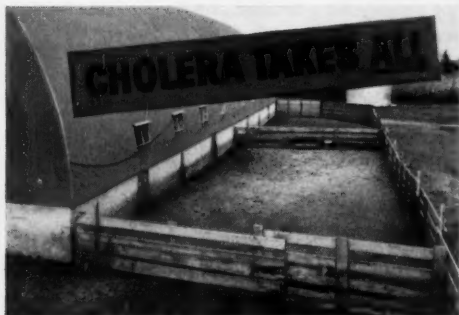
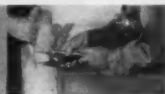
M·L·V

The original modified live virus hog cholera vaccine

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consult your
veterinarian

he knows best—what's best



You risk not only low-priced pigs but all the corn, care and cash you've put in them!



consult your
veterinarian
*he knows best—
what's best*

If you lost just a few pigs or a slim profit—maybe you could gamble with cholera. But cholera takes more—the pigs, the profits, plus all the corn, care and cash you have in them! Cholera hits peaks in late summer or early fall, and in spring. Just when most farmers have the most investment in their hogs. Compared to possible losses, vaccination is cheap. It's your only protection!

Cholera struck on many hundreds of farms in 1959. That it didn't strike more times is to the credit of vaccination—in large measure to professional vaccination with M.L.V. In the last 9 years, thousands of veterinarians have protected millions of swine with Fort Dodge M.L.V. Used simultaneously with serum, M.L.V. provides immediate and prolonged immunity.

M·L·V

The original modified live virus hog cholera vaccine

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urge the hog farmer
to use professional
vaccination

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M·L·V T.M.

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For many years Friskies Meal has stood for the finest in a dry dog food. Now a great new Friskies Meal has been developed... finer even than the Friskies Meal you may already know!

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Great new convenience! New Friskies Meal mixes as a dry dog food should—instantly...completely—without caking or mushing—ever!

There's no sticking to bowl—never a messy cleanup after feeding.

Complete, balanced formula! As always, Friskies Meal is complete with every vitamin, mineral and other nutrient dogs are known to need. In addition, new Friskies Meal is high in digestibility.

Recommend new Friskies Meal today. It's everything dog owners could want in a dog food—just what you'd expect from

Carnation.



For variety... suggest Canned Friskies—hearty with meaty-rich flavor—and Friskies Cubes—entirely new kind of dog food so palatable it can be fed dry.

quietly walked up the ramp...



*"A gelded Welsh pony refused to walk up a loading ramp to the truck. Half an hour was wasted in applying ropes, pulling, pushing, and encouraging with oats, during which time the pony accidentally lacerated a lip. Within ten minutes after it was given 150 mg. of the drug [SPARINE] intravenously, the pony quietly walked up the ramp on the first attempt."**

Wyeth Laboratories Philadelphia 1, Pa.

*Gorman, T.N.: J.A.V.M.A. 134:564
(May 15) 1959.

AVAILABLE:

TABLETS: 25, 50, 100 mg., vials of 50.

INJECTION: 50 mg. per cc., vials of 10, 30,
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**Endotracheal
Intubation**



Recognizing the possibility of improving important instruments, Davol's Research and Development group has again collaborated with members of the medical profession—anesthesiologists in this case—and have introduced the NEW TRANSPARENT ENDOTRACHEAL TUBE. The so called "blind spot" that so often obscures important landmarks during intubation is no longer a problem. The exact positioning of the distal tip is now possible. Presence of excessive secretions as well as types of secretion—sanguineous, serous or mucoserous—become visually apparent. Trauma due to pressure of Endotracheal Tube, during prolonged surgical procedures, may be minimized by using these relatively thermolabile tubes.

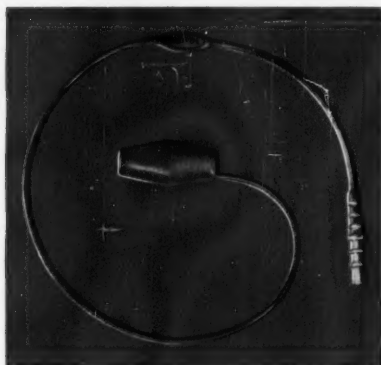
with New Davol

TRANSPARENT

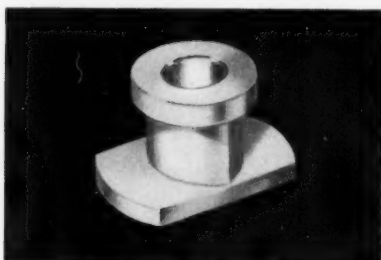
Endotracheal Tubes*

- Can be sterilized by all usual methods.
- Unobstructed field of vision during intubation.
- Obstructive secretion may be aspirated under direct vision.
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with Inflatable Cuffs with pilot balloon



**with Keown Endotracheal Tube Guard
(MOUTH PROP)***

Again, to fill a long existing need for the anesthesiologist Davol has designed an Inflatable Cuff incorporating those suggestions most frequently submitted to our Technical Staff:

(1) Symmetrical inflation - from 3 cc. to 15 cc.

(2) Pilot balloon - providing (a) visual insurance of inflation in situ (b) sensitive to the extent that an intelligent estimation of intracuff pressure can be made by palpation (c) nylon connector in proximal end of inflating tube permits use of either slip or Luer-lok Syringe without needle.

(3) Markers indicating range of sizes of tubes that each cuff will fit are placed on each pilot balloon.

Designed to provide a means of eliminating possible occlusion of the tube due to compression by the patient's teeth and lessens the possibility of the Endotracheal Tube's kinking in the mouth. Will accommodate all sizes of Endotracheal Tubes up to 42 French Scale.

**By personal communications.*

These specialized instruments for Endotracheal Intubation are the result of a continuous effort to keep abreast of the latest advances in this important field.

Your surgical supply dealer will be glad to furnish you with detailed information on styles and sizes of Davol Endotracheal Tubes, or write Davol Rubber Company, Providence 2, R. I.





FROM THE AVMA WASHINGTON OFFICE

J. A. McCallam, VMD
Brig. Gen. USA (Ret.)

LEGISLATIVE

Kansas City Urges Increased Brucellosis Funds

Kansas State Legislature adopted their House Concurrent Resolution 7, requesting U.S. Congress to appropriate funds for continuation of the brucellosis eradication program on an active and progressive basis in keeping with assurance of federal cooperation when the program was offered the states. The resolution was printed in *Congressional Record*, Feb. 8, 1960, at request Sen. Schoppel, Kan.

Sen. Aiken Attacks Brucellosis Fund Cutback

In a Senate speech (Cong. Rec., Feb. 11, 1960), Sen. Aiken (R., Vt.) attacks "penny wisdom and pound foolishness" in the government operation of brucellosis eradication program. Deceleration of program resulting from cutbacks in 1960 fiscal funds (see JOURNAL, July 1, 1959, adv. p. 14) is being reflected in slow-down of progress, he says. Sen. Aiken calls for restoration of funds to at least the 1959 level (\$20 million) or it will, in long run, cost an additional 71 per cent to accomplish the objectives of the program. The Senator pointed out the sooner the disease is eradicated the sooner producers will be free of a \$25 million loss annually, and that early eradication is in the interest of public health (this connection see JOURNAL, March 1, 1960, adv. p. 10).

Pacific Animal Quarantine Station Requested

A resolution adopted by Oregon State Board of Agriculture, presented to the Senate by Sen. Morse (Cong. Rec., Feb. 15, 1960), asks Congress for funds for the establishment and maintenance of an animal quarantine station to serve the Pacific area. No satisfactory federal facilities exist for holding and examining foreign animals such as provided on Atlantic seaboard.

Keep Writing for Self-Employed Tax Deferment Bill

Indications are that the Senate Finance Committee will report favorably on H.R. 10, self-employed tax deferment bill (probably with amendments). This encouraging news, based on remarks by Sen. Smathers on senate floor Feb. 18, believed due to change in treasury position regarding Smathers-Morton-Keogh-Simpson bill. **Do not be complacent**—bill still must pass Senate! All interested, continue writing your senators to support this measure.

NEW BILLS

Sen. Bennett (R., Utah) submitted amendment Feb. 11 to S. 2086, National Wildlife Disease Laboratory bill (see JOURNAL, July 1, 1959, adv. p. 14) to provide for the laboratory at any state-owned university having one or more existing programs useful in carrying out provisions of the Act. Initial

(continued on adv. p. 12)

AN EFFECTIVE, PRACTICAL, SAFE PREVENTIVE FOR SWINE ERYSIPELAS

Affiliated Brand

ERYSIPELAS BACTERIN

AFFILIATED LABORATORIES CORPORATION

White Hall, Illinois

The National Laboratories Corp.

The Gregory Laboratory, Inc.

Grain Belt Supply Co.

Corn Belt Laboratories, Inc.



Product is available to graduate veterinarians only.

Washington News—Continued

NEW BILLS

bill provides laboratory be established at land-grant college or university having a school of veterinary medicine.

H.R. 10255, Rep. Fogarty (D., R.I.), amend PHS Act or provide federal assistance to states which award scholarships to medical and dental students.

S. 3007, Sen. Clark (D., Pa.) and 21 co-sponsors, to authorize federal loans to colleges and universities for construction, improvement, etc., of classroom buildings and other academic facilities.

H.R. 10330, Rep. Dingell (D., Mich.), amend Internal Revenue Code 1954 to prohibit deductions of certain expenditures as trade or business expenses; companion bill to *S. 2040* (See JOURNAL, July 1, 1959, adv. p. 14).

H.R. 10341, Rep. Harris (D., Ark.), amend PHS Act to authorize grants-in-aid to universities, hospitals, laboratories, and other public or nonprofit institutions to strengthen programs of research and research training in sciences related to health.

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One that will surely hit the spot with your dog(s)
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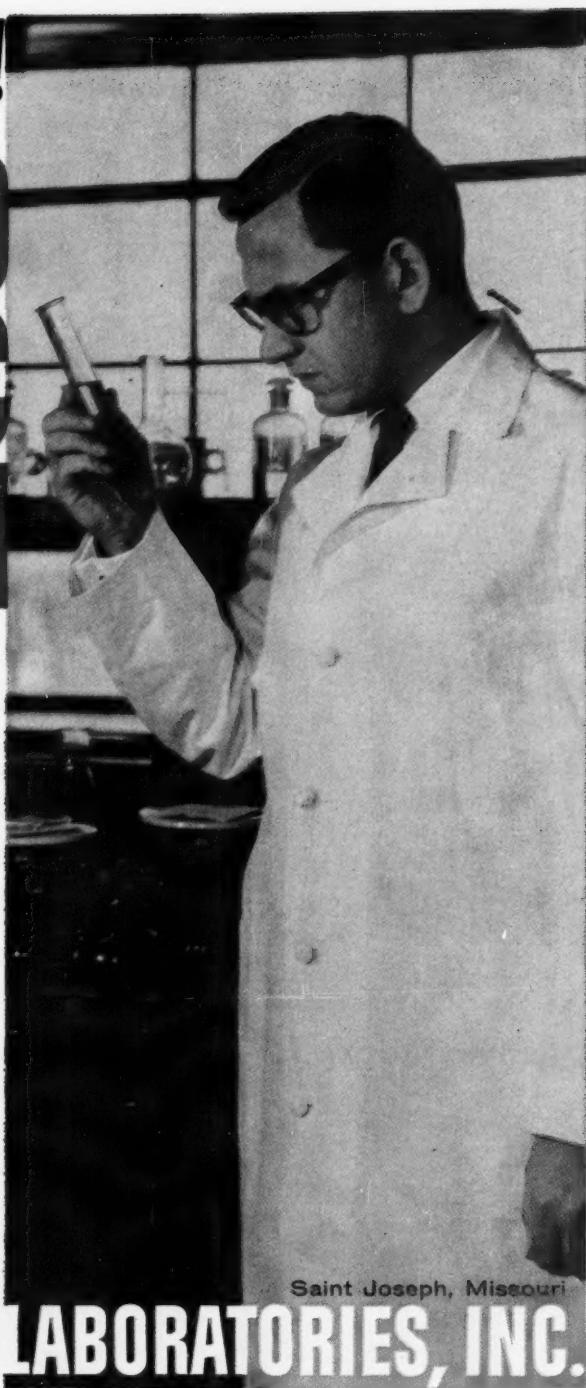
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**\$50 Million a Year
Is Lost by Farmers**

For this reason, organizational

MANY types of scours hit cattle, hogs, and sheep. Especially watch young animals, favorite targets of the disease. An ever-present problem, it's worse in late winter and spring. And in addition to being a livestock disease, it's also a symptom of other diseases. The American Foundation for Animal Health says, "An early diagnosis is important when signs of scouring first show up."

Some forms of scours are not contagious, but others spread rapidly. Animals showing signs of scours should be isolated as well

Hidden killers which live on for years in the soils of pastures and barnyards are costing farmers over \$200 million a year in livestock losses.

That report issued by American Foundation for Animal Health, cautions national owners that these disease organisms of the "coccidia" group cause their greatest losses during summer months but ahead.

**Beware Of
Erysipelas
In Your Hogs**

This spring, farmers need to be more alert than ever against losses caused by swine erysipelas, according to a report issued today by the American Foundation for Animal Health.

This disease has now become the No. 1 swine problem in some states, the report says. Nationally, it ranks second only to hog cholera, and causes farm losses of over \$25,000,000 per year. The problem has grown to such an extent that it has been singled out as this month's prime target for National Livestock Loss Prevention Year.

Farmers were cautioned to watch for such symptoms as prostration, weakness, loss of appetite.

Threat to Cattle
Biting, northerly winds which force cattle into barns for the winter also usher in the danger season for winter dysentery of cattle, the American Foundation for Animal Health warned today.

This disease may affect horses and dairy cattle housed during the months of November to March. Veterinary medical authorities said that while winter dysentery may spread rapidly through a herd, causing substantial economic loss, the actual death rate is very low.

Winter scours, as the disease is sometimes called, appears suddenly in many cases only one animal shows symptoms at the outset, but the disease quickly spreads to other animals.

Time to Check Swine Against Brucellosis

This is the season when hog should be taking steps to elude the hazard of brucellosis in their herds, according to a bulletin today by the American Farm for Animal Health.

five steps, veterinarians said, is to make certain that pigs purchased for breeding spring pig crop are free of disease, which is similar to brucella.

The Foundation suggests that farmers buy boars only from known sources to be free of the disease. The Foundation says that is not possible, the Food and Drug Administration urged farmers to have a veterinarian inspect newly-purchased boars for signs of disease. The boars show distinct signs of disease, separate from the home herd for 30 days and then tested again for disease, and in a breeding pen.

This spring, farmers need
be more alert than ever against
the danger caused by swine erysipelas,
according to a report issued
yesterday by the American Foundation
for Animal Health.

The disease has been the top swine problem in 11 states, the report says. Nationally it ranks second only to hog cholera and causes farm losses of over \$25 million per year. The problem has grown to such

...watch for such symptoms as inactivity, weakness, loss of appetite, a stiff gait, or a hunched posture in the quarters. Early diagnosis is important.

It is to be chosen where a
In areas where a
become a problem, a
considered a hard v
crises. The selection
gram and the type
be used should be
erine! In buying
animals, farmers sh
record of the
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new stock for 30 d
disease a chance
are, farmers should
They late path record of the
which they come
infuse new stock for 30
to give better disease

Hidden killers which live on for years in the soils of pastures and barnyards, are now costing farmers over \$30,000,000 a year in live-

That report, issued by the American Foundation for Animal Health, cautioned animal owners that these disease organisms of the "clostridial" group cause their greatest losses during summer months just ahead.

These killer organisms are the cause of such livestock diseases as lockjaw, blackleg, malignant edema and enterotoxemia. The organisms generally enter the animal while grazing or through wounds. Losses are so heavy from these causes that authorities have urged clostridial infection as the subject of a special Joint Report of National Livestock Prevention Year.

Preventive vaccination done against none of these

This spring, farmers must be more alert than ever against losses caused by swine erysipelas, according to a report issued today by the American Foundation for Animal Health.

This disease has been the No. 1 swine problem in so many states, the report says. Nationally, it ranks second only to cholera, and causes farm losses of over \$25,000,000 a year. The problem has grown so serious that it has been declared a national health hazard as this month's National Livestock

Farmers were
watch for such
prostration, weak
petite, stiff gait,
to remain in quai
diagnosis is impo

Stockmen Fear New Outbreak Of Cholera

Livestock authorities are seriously concerned this Spring about the possibility of a new wave of hog cholera sweeping the nation's farms.

The disease may cause fever and an estimated 500,000 pigs annually. There are different types of swarming agent. State health authorities said that determining of which to use depends on condition of the pigs, their age and local circumstances should be determined by a veterinarian.

The virus of hog cholera is most easily known to veterinarians. A drop of the disease would start an epidemic.

About this time of year, "Time to buy" more and more frequently. "Time to buy" your car, the service station signs say. "Time to buy" your down, caulking, insulation, etc., and "winter" building material men

[illegible]

Disease The following nature of the
Cattle spreading nature of the

Now That Cattle Are In Danger High

Butting, northerly winds which force cattle into barns for the winter also usher in the danger season for winter dysentery of cattle, the American Foundation for Animal Health warned today.

Adventist authorities are
heavily concerned this spring
the possibility of a new
big cholera sweeping the
tarns.

have an...
...to a...
...the disease...
...the American Foundation...
...Health. This may...
...stage for fresh outbreaks...
...search for heavy...
...the Found...

so that a veterinarian can be called promptly to make a diagnosis and the treatment.

The most prominent symptom winter dysentery is acute. disease does not affect the mal's appetite in all cases. In severe cases abdominal pain may be evidenced by twitching of the skin.

Alert Farmers To Hog Disease

This disease has now become the No. 1 ovine problem in the states, the report says. National Animal Health.

problem has grown to such an extent that it has been singled out as this month's prime topic for the National Livestock Loan Pledge Year.

Farmers were cautioned to watch for such symptoms as a sudden weakness, loss of appetite, a sick gut, or a tendency to strain in quarters. A prompt remedy is important if an outbreak is to be controlled.

In areas where crystal disease has become a problem, and where the disease has been recommended as a herd vaccination program. The selection of a vaccine and the type of vaccination should be left to the

Hidden Killers Cost Farmers \$20,000,000!

Hidden killers which live on for years in the soils of pasture and barnyards, are now costing farmers over \$20,000,000 a year in livestock losses.

That report, issued by American Foundation for Animal Health, contained animal owners the "clandestine" group cause their greatest losses during summer months just ahead.

Hidden Killers In Soil Cause Livestock Losses

These killer organisms are the soil-borne, blackish, malignant disease, which causes the animal to die generally under the animal's grazing or through wounds.

These killer organisms are the soil-borne, blackish, malignant disease, which causes the animal to die generally under the animal's grazing or through wounds.

Thousands of newspaper articles, like these, stressing the value of veterinary service. Thousands more in farm magazines, plus radio, television. Underwritten by these member companies . . .

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- The National Laboratories Corp.
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Soil Organisms Cause Animal Loss

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Worry About New Wave of Hog Cholera

Authorities are seriously concerned this spring with the possibility of a new wave of hog cholera sweeping the nation's farms.

The American Foundation for Animal Health says farmers in certain areas let down their guard by permitting pigs to go unvaccinated against the disease.

Alert Farmers To Hog Disease

This year, farmers need to be more alert than ever against disease caused by swine erysipelas, according to a report issued today by the American Foundation for Animal Health.

This disease has now become a No. 1 swine problem in some states, the report says. Nationally, it ranks second only to hog cholera, and causes farm losses of over \$20,000,000 per year.

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Baby Pig Problems Run Into Big Money

BABY PIG problems cost farmers of this country nearly a half-billion dollars a year, with the three worst killers being anemia, scours and TGE (transmissible gastro-enteritis).

The toll of baby pig diseases is so great that a special effort will be made in February by the National Livestock Loss Prevention Year program. There will be emphasis on how to take preventive steps against occurrence of disease.

Anemia is the result of deficiencies of iron and copper in the ration of young pigs, according to the American Foundation for Animal Health, Des Moines.

Other methods include painting a sow's udder in a pen. Iron can be caused by a iron, or brought on by iron. However, pigs chilled.

Warning On Hog Disease

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Danger Season For Cattle Dysentery

Blowing, northerly winds which force cattle into barns for the winter also usher in the danger season for winter dysentery of cattle, the American Foundation for Animal Health warned today.

This disease may affect both beef and dairy cattle housed during the months of November to March. Veterinary medical authorities said that while winter dysentery may spread rapidly through a herd, causing substantial economic loss, the actual death rate is low.

Soil Organisms Cause Animal Loss

Hidden killers which live on for years in the soils of pasture and barnyards, are now costing farmers over \$20,000,000 a year in livestock losses.

That report, issued today by American Foundation for Animal Health, contained animal owners the "clandestine" group cause their greatest losses during summer months just ahead.

Ten Point Plan For Winterizing Livestock Given

A 10-point plan for "winterizing" livestock was recommended to farmers today by the American Foundation for Animal Health.

Highlights of the plan, designed to provide better winter health and production are:

1. Check sanitation and ventilation facilities of hog and poultry houses to guard against pneumonia and other respiratory diseases.
2. Exercise caution in allowing cattle into draft-crippled fields of corn, beans, grass or clover, whose seedlings may result.

Farmers Are Alerted On Swine Problems

That report, issued today by American Foundation for Animal Health, contained animal owners the "clandestine" group cause their greatest losses during summer months just ahead.

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Farmers Urged To Watch For Brucellosis

FARMERS URGED TO WATCH for "brucellosis," the most dangerous of diseases which can be passed from one animal to another, according to a report issued today by the American Foundation for Animal Health.

This disease has now become a No. 1 swine problem in some states, the report says. Nationally, it ranks second only to hog cholera, and causes farm losses of over \$20,000,000 per year.

That report, issued today by American Foundation for Animal Health, contained animal owners the "clandestine" group cause their greatest losses during summer months just ahead.

Shipping Fever Takes Toll

Farmers on this time of year need to watch out for a fall disease, "shipping fever," which may cost them heavily.

This disease has now become a No. 1 swine problem in some states, the report says. Nationally, it ranks second only to hog cholera, and causes farm losses of over \$20,000,000 per year.

That report, issued today by American Foundation for Animal Health, contained animal owners the "clandestine" group cause their greatest losses during summer months just ahead.

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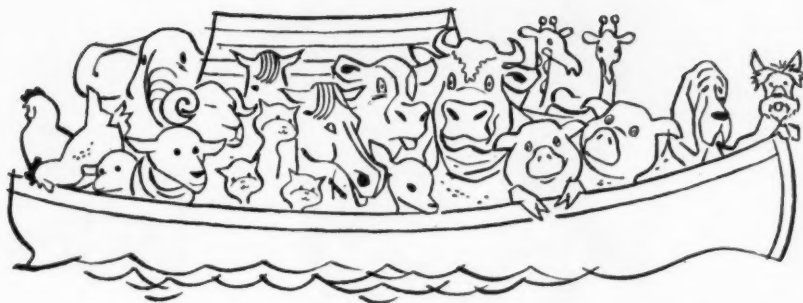
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DARIBIOTIC Soluble—convenient oral administration in liquids for intestinal infections.

*U.S. Patent No. 2,565,057

1. Barr, F.S., Carman, P.E. and Harris, J.R.: Synergism and Antagonism in Antibiotic Combinations; Antibiotics and Chemotherapy; 4:818 (1954).

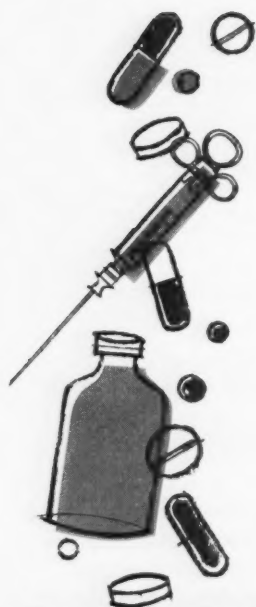
2. Baker, W.L.: Clinical Use of Injectable Neomycin and Polymyxin B; Veterinary Medicine, 53 (1958):275.

3. Barr, F.S., Harris, J.R. and Carman, P.E.: Intramuscular Treatment of Staphylococcal Mastitis with Neomycin Sulfate and Polymyxin B Sulfate; J.A.V.M.A., 132 (1958): 110.



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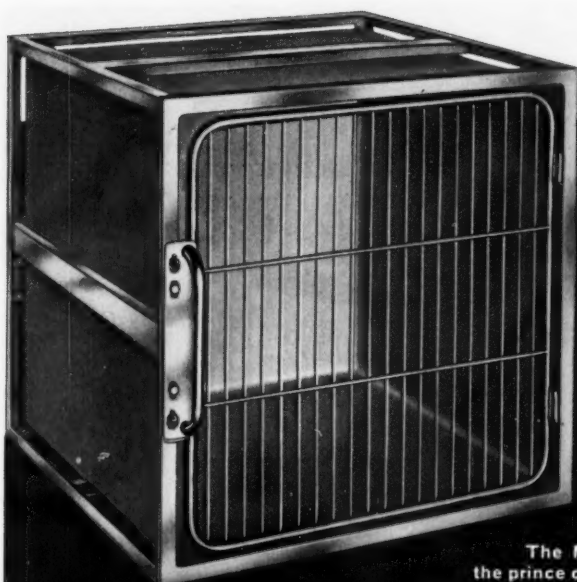
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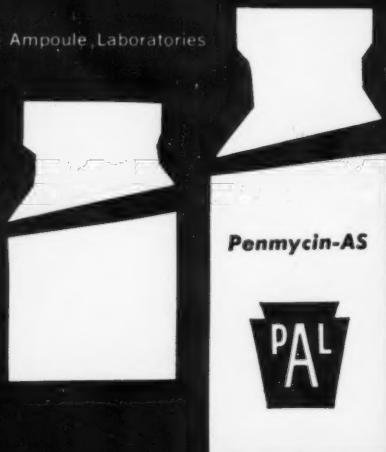
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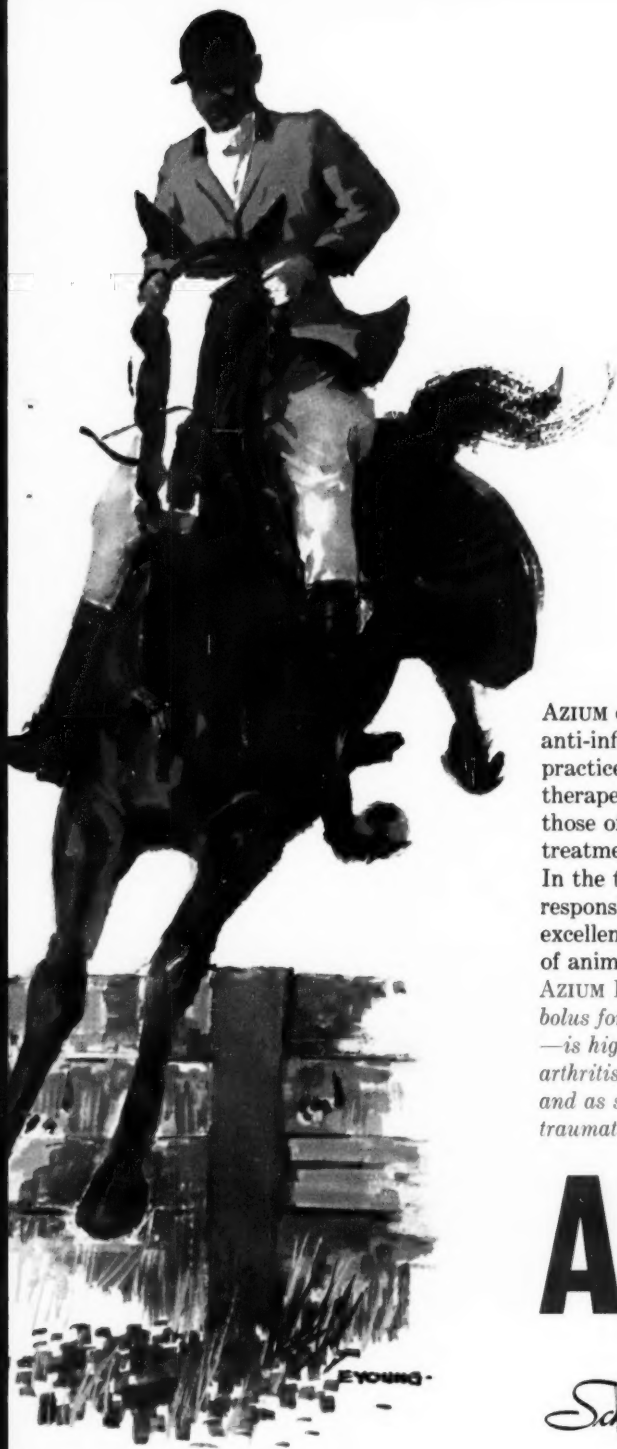
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1. Kakavas, J. C., et al.: J. Am. Vet. M. Ass. 119:203 (Sept.) 1951.



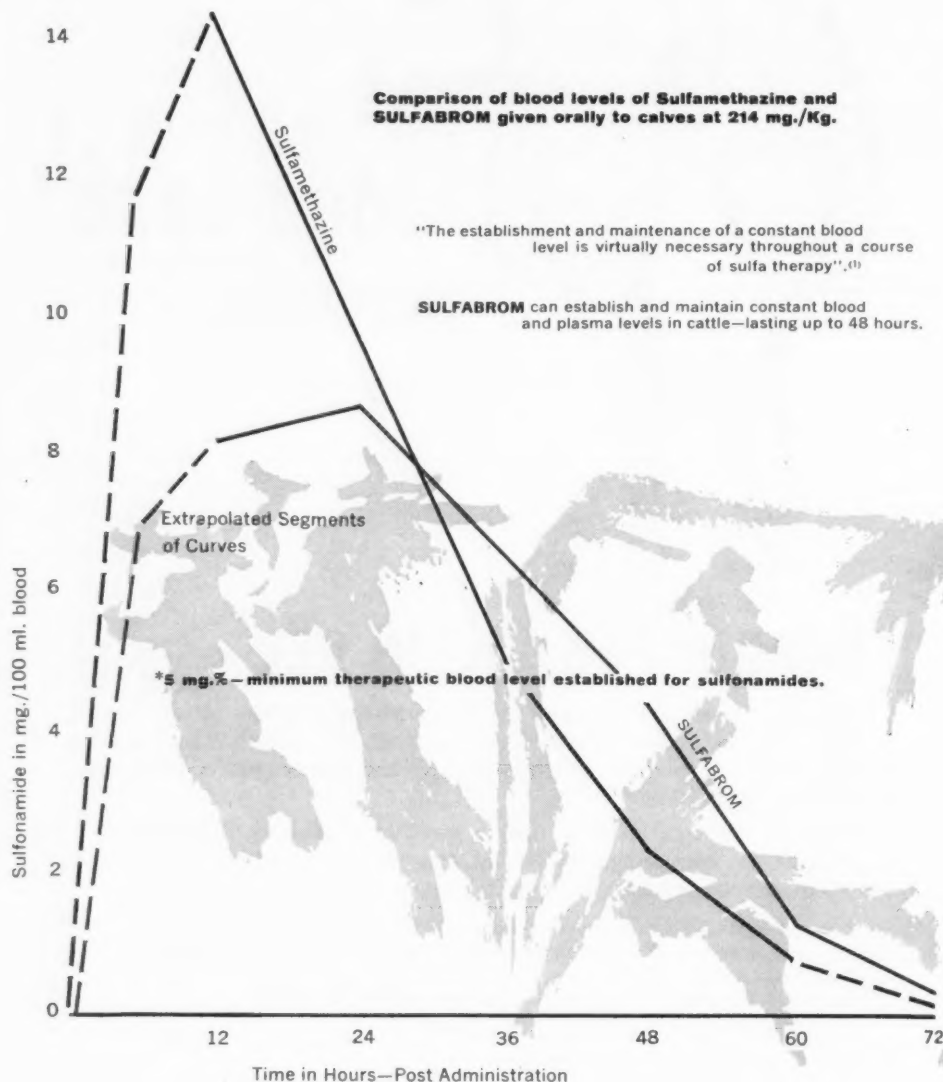
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(1) Stowe, C.M. et al.: *Am. J. Vet. Res.* **19**:345 (April) 1958.

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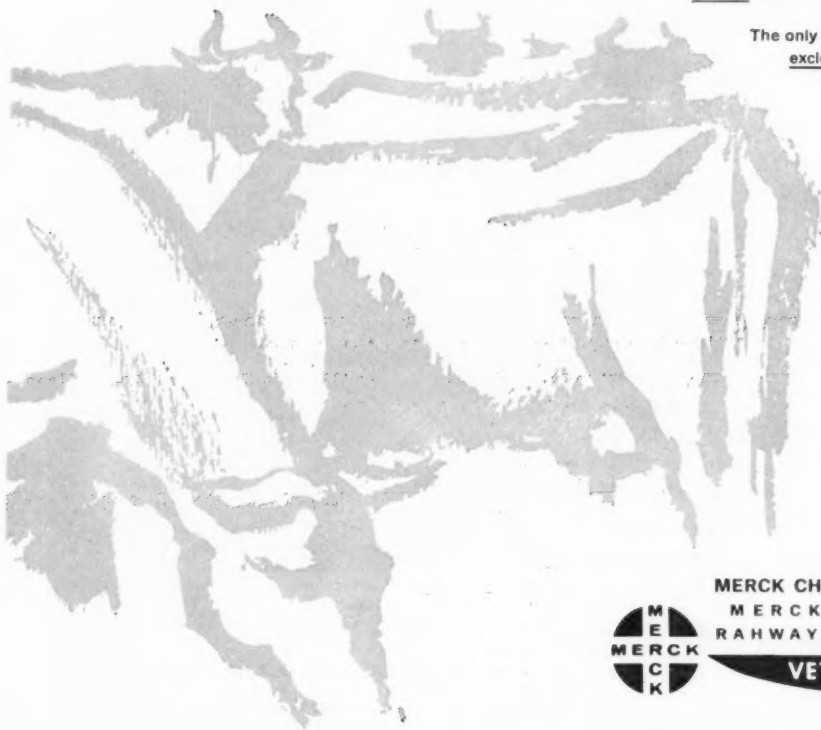
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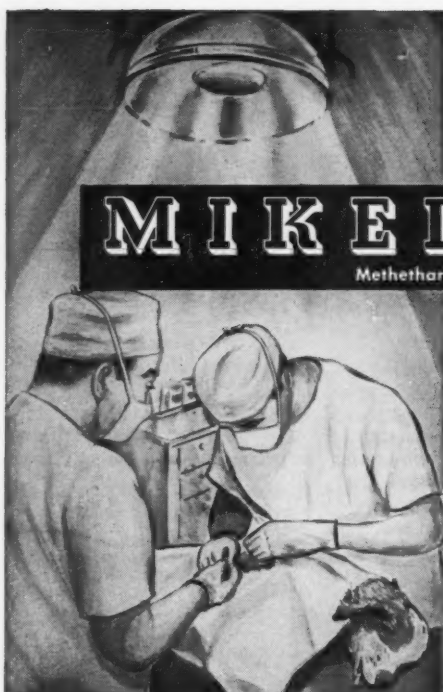
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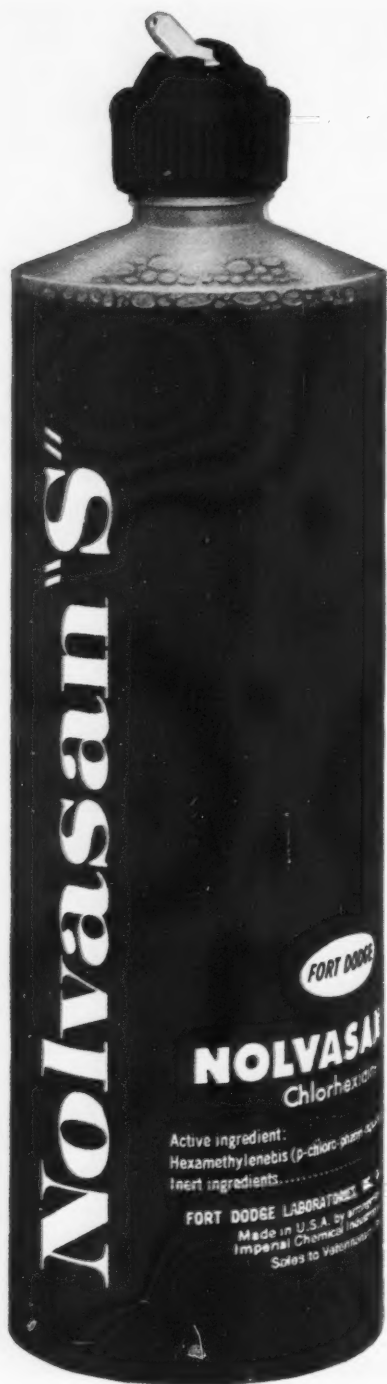


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New Concepts of

Leptospirosis

in Animals

Erskine V. MORSE, D.V.M., PH.D.

VETERINARIANS, physicians, and public health officials have been intensely concerned with leptospirosis for over a decade. Justifiable interest stems from these facts: (1) Leptospire have a wide mammalian host range including man; (2) these infections are no longer considered exotic, for all serotypes have potential worldwide distribution; (3) significant economic loss to livestock owners is evident; and (4) these infections are not decreasing in our animal and human populations. It would be difficult to discuss adequately all leptospiral diseases. The material presented will deal, unless otherwise indicated, with *Leptospira pomona* infections in the United States.

Leptospira pomona is the infecting serotype in 98 per cent of the enzootics in cattle and swine in the United States. Serologic surveys indicate that 3.5% to 11.2 per cent¹⁰ of the cattle, and 37 to 22 per cent¹³ of the swine have experienced infection. It

is estimated that 2 to 4 per cent of these livestock are actively infected. Combined losses in cattle and swine probably exceed \$200 million annually.

A major problem confusing the serodiagnosis of leptospirosis has been the presence of agglutinins other than those for *L. pomona* in domestic livestock. *Leptospira sejroe*,^{19,23,54} *Leptospira canicola*,^{5,19,50} *Leptospira icterohaemorrhagiae*,^{5,19,33,50} *Leptospira autumnalis*, *Leptospira hebdomadis*, and *Leptospira grippotyphosa*⁵⁰ antibodies have been detected in the serums of cattle or swine in the United States. These findings may be the result of: (1) clinical or, more probably, subclinical dual specific serotype infections past or present; (2) cross reactions of a specific nature, i.e. antigens in common shared by 2 or more serotypes; (3) antigens shared with other bacterial genera;⁴⁷ or (4) nonspecific agglutinins which result in false positive reactions. The isolation of *L. pomona* from a cow having a 1:600 *L. sejroe* serum titer but negative for *L. pomona*,²³ and from a dog which was seropositive at 1:400 for *L. autumnalis* and 1:100 for *L. pomona*³⁹ presents some intriguing possibilities. The ex-

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act causes of serologic cross reactivity remain obscure. The phase of infection,^{9,28} the host species involved,^{28,53} and individual differences within the species^{28,53} are all contributing factors in the leptospiral agglutination cross-reaction phenomena. A more complete understanding of this important diagnostic problem is critically needed.

An unfortunate misconception regarding bovine and porcine leptospirosis has arisen in some areas. The family physician, upon learning that his patient's cattle or swine have leptospirosis, concludes that the cause is the Weil's disease agent, i.e., *L. icterohaemorrhagiae*. This is understandable, but nevertheless the ensuing confusion may lead to unnecessary quarantines and needless concern. While cattle^{3,17,26} and swine^{18,40} have been naturally infected with *L. icterohaemorrhagiae*, one should emphasize that this is unusual, and the vast majority of infections are due to *L. pomona* in the United States. However, because of the broad host range for *Leptospira*, we should always be prepared for the possibility of invasion in our domestic livestock by other serotypes. Serologic surveys suggest that this has probably occurred in some instances although not proved by actual isolation and definitive identification of the agent.

Leptospirosis is increasing in prevalence. Bovine and porcine *L. pomona* infections have not significantly decreased in Iowa during 1955 to 1958 according to one writer.⁵ It would appear natural that similar trends continue in other agricultural areas. Leptospirosis in man is either being recognized more readily or it is on the increase in Iowa. From 1951 to 1957, this state reported 5 cases, while in 1958 more than 15 cases were recognized.²⁷ Indications were that "almost all" were due to *L. pomona*. Leptospirosis continues as one of our major livestock disease problems. Critical appraisal of the situation does not indicate that its prevalence is decreasing in farm livestock.

Specific antigens common to leptospires and brucellas do not appear to exist.²³ Therefore, the use of *L. pomona* bacterin should not influence *Brucella* serum agglutinins for animals vaccinated with strain 19.³¹ Reports have been circulated that cattle previously classed as "negative" on the brucellosis test became "reactors and suspects" following vaccination with

L. pomona bacterin. Circumstances pertaining to such findings warrant investigation and a thorough study of the phenomena is indicated. From a practical standpoint, tests for leptospirosis and brucellosis do not appear to be jeopardized by serologic cross reactivity. Progress in some brucellosis control programs has been hampered to a minor degree because of the presence of leptospirosis. Dairymen have been reluctant to lose brucellosis accreditation during "abortion storms," which they wrongly guessed were due to brucellosis. Later the trouble was found to be *L. pomona* infection. It is of the utmost importance that leptospirosis is not confused with brucellosis in either animals or man.

No single factor alone is responsible for the severity and prevalence of leptospirosis in livestock. Some major influences which interact in perpetuating infection are: (1) the domestic animal hosts involved, (2) wildlife reservoirs; (3) general husbandry practices; (4) environmental conditions; and (5) virulence of the infecting serotype.

Domestic Animal Hosts

The ideal host for perpetuating *L. pomona* infection is a species which maintains a urinary shedder state of long duration and eliminates great numbers of leptospires in urine. Swine fulfill both these criteria, while cattle follow closely. Ruminants, and swine to a lesser and variable degree, which normally maintain a urinary pH above 6, favor transmission of leptospires. In theory, at least, pH of urine may directly influence duration of the carrier state and effective transmission of leptospires to new susceptible hosts. In cattle and swine, the greatest numbers of leptospires are excreted in urine approximately 25 to 35 days following subcutaneous exposure. During this period, direct darkfield microscopic examination of the urine is most likely to yield positive results. Very few, if any, organisms are eliminated prior to 10 days by most experimental hosts. Sheep³⁶ and dogs¹⁵ remain carriers for approximately 60 days, but usually few leptospires are excreted in their urine. They do not appear to be highly effective in transmitting *L. pomona* infection. Horses ap-

parently do not shed great numbers of leptospires in the urine^{8,11} and probably are not significant spreaders of leptospirosis. Goats shed *L. pomona* for approximately the same periods as do sheep but, in contrast, goat urine may contain numerous leptospires.³⁵ Caprine leptospirosis, it is theorized, represents a greater threat to susceptible animals than the disease in sheep, horses, or dogs.

In this country, it appears that severity of and mortality due to leptospirosis has been greater in beef type cattle than in the dairy types. Icterus, prostration, and death are attributed to leptospiral disease in beef animals in Iowa. The etiology of this condition is not definitely known, and it is questionable whether severe manifestations are due to *L. pomona*. Limited experimental evidence indicates that grade Hereford calves were more seriously affected than were Holstein-Friesian calves of approximately the same age and raised under identical conditions.³⁸ Critical investigations of susceptibility in beef as compared with dairy cattle, as well as comparative pathogenesis of *L. pomona* infections in the two types of cattle, are warranted.

Wildlife Reservoirs

The exact status, extent, and domestic animal health significance of *L. pomona* infections in wild mammals is not thoroughly understood. Surveys indicate that serum antibodies are present in many wildlife species in the United States.^{20,30,49} *Leptospira pomona* has been isolated from a deer,⁴⁴ an opossum,⁴⁶ a wildcat,³⁰ and skunks³⁰ in the United States. Deer have been experimentally infected.⁵⁷ The degree to which these ruminants will transmit the disease is not known.

Two possibilities exist regarding the importance and role of wildlife species as hosts for *L. pomona*. First, as leptospirosis decreases in farm livestock, the wildlife population will become the significant source of infection for man and other animals. Second, as *L. pomona* infections become less prevalent in farm stock, the same trend will occur in wildlife hosts. This supposition is based on the theory that farm animals are actually serving as the perpetual sources of *L. pomona* infec-

tions in deer, bobcats, etc. In other words, our infected cattle and swine are the important reservoirs of leptospirosis for wild animals and not vice versa.

Isolation and identification of infecting serotypes are imperative. Such data will provide needed information as to the magnitude and scope of the problem and serve as a future guide for control measures.

The possible role of arthropods in the transmission of leptospirosis should not be overlooked. Infected wild hosts may provide leptospires for ingestion by vectors, and thence transmission to livestock may occur. It has been reported that *L. pomona* will persist for 518 days in the tick, *Ornithodoros turicata*.¹² Experimental transmission to a susceptible host was still possible 232 days following ingestion of leptospires.

General Husbandry Practices

Agricultural management and general husbandry practices in an area contribute to incidence and probably severity of leptospirosis. The hog wallow, muddy water hole, overcrowded feedlot, occasional flooding of pastures, lack of mangers and feed bunkers, and intermingling of cattle and swine are obvious and paramount factors for perpetuating leptospirosis.

In Iowa, during 1958, the highest seasonal incidence of bovine leptospirosis occurred during June through November.⁵ At this time, cattle were on pasture, and water was readily obtainable from small streams or waterholes. Water-borne leptospirosis outbreaks would be expected to reach maximal incidence at this time; yet porcine leptospirosis had its lowest reported incidence during this period. However, the cardinal sign of the infection in swine is abortion, and there are relatively few pregnant sows in summer. The greatest number of positive serum-agglutination tests were found during spring and fall prior to or concurrent with the farrowing seasons. Man's manipulation in order to have fall and spring pig crops appears to influence the observed incidence of *L. pomona* infections.

Feeds will influence, within limits, the ability of individuals to transmit leptospirosis. Swine rations which are rich in animal protein or contain high levels of

minerals, salts of weak bases, and strong acids will tend to maintain urine at pH 6 or below. Such a reaction is unfavorable to *L. pomona* and probably is a factor in determining the duration of the carrier state and the number of viable organisms shed by swine.³⁴ The diet then may also be a factor in the wide variations reported for the urinary shedder state in swine^{34,40} and other livestock as well.

The widespread use of additives in livestock feeds and the effect upon leptospiral infections is a matter of conjecture. It has been reported¹ that serologically negative mice harbored *Leptospira ballum*. Other investigators have isolated leptospires from serologically negative animals.¹ *Leptospira pomona* was present in kidneys of experimentally infected hogs which were fed feed containing chlortetracycline,^{24,32} but serum titers were not demonstrable at slaughter. While results were not conclusive, and since kidney infections without concurrent serum titers can occur with nonmedicated rations, a critical study of this situation as it relates to various nutritional regimens would seem warranted.

Successful prophylaxis, treatment, and control measures might be instituted simultaneously by feeding rations favoring acid urine and containing tetracycline antibiotics, and by vaccination. The combination of 3 approaches might prove to be more efficacious than any single therapeutic or prophylactic measure alone.

Purchase and continual influx of feeder and replacement stock will increase the probability of introducing leptospiral diseases. The livestockman who maintains a "closed herd" with minimal additions will have avoided one of the most potent factors in disseminating the disease. Frequent additions and replacements to the herd are known to be the source of many severe epizootics in both cattle and swine.

Environmental Conditions

Water-borne sources of leptospirosis represent a major hazard. Epizootics in animals and man continually stress this fact. Rainfall, humidity, and temperature climate provide the environment which best favors transmission and establishment of leptospirosis in a region. Extremely cold weather, especially when as-

sociated with slow freezing and thawing, is detrimental to the extrahost survival of *L. pomona* and is independent of pH.⁴² Ovine leptospirosis is not uncommon in New Zealand, while it is rare or unrecognized in the United States.⁴⁴ In New Zealand,²² lush, wet pastures, alkaline soil, as well as temperature climate, provide suitable enzootic conditions. In the United States, sheep pastures are generally on dry marginal lands, and climatic conditions in the range states present extremes. Such a grazing area is less favorable to leptospiral survival and transmission.

The severity of leptospirosis in livestock as reported in various geographic areas is no doubt attributable in some degree to differences in virulence for respective strains of *L. pomona*. Mortality has been higher, and clinical manifestations, i.e., icterus, anemia, and hemoglobinuria, have been dramatic for bovine enzootics in western range states as compared with those in predominantly dairying areas. Variations in virulence for a single strain and between strains have been observed. Cultivation in mediums leads to a decrease in virulence according to most reports.^{14,16,29,52} Serial passage of *L. pomona* in embryonating chicken eggs rendered the agent less pathogenic for cattle.⁴³ Conversely, increased virulence has been reported for 2 *L. pomona* strains following propagation in mediums.² Serial passages in laboratory animals have been found to increase pathogenicity.^{21,41,51,55} Other workers have found little alteration in virulence following repeated transfer in animals.^{25,45} It would seem that the topic of virulence warrants appraisal and should be closely correlated with a thorough study of mutation or variation among the leptospires. The hemolysin of the leptospires represents a known factor which operates in the virulence mechanism. Although the exact nature and mode of action *in vivo* remains obscure, it has been reported that hemolysin resembles an enzyme and activity parallels that of *alpha* toxins of clostridia organisms.²⁵ It has been found that a "supersonic extract" of *L. icterohaemorrhagiae* elicited an intracutaneous response in guinea pigs following injection.⁴⁵ The investigators interpreted this as evidence of a toxic principle other than hemolysin.

The leptospires are of considerable consequence and represent a serious health

problem in our domesticated animals and house pets. Similar hazards exist for our wild fauna and certain occupational groups. The economic and public health aspects of leptospiral infections have created common problems for the cooperative efforts of livestockmen, veterinarians, public health workers, and physicians. All groups have expressed concern and interest in developing an effective program for the control of leptospiral infections. The various segments of the livestock industry should cooperate effectively in decreasing the incidence of leptospirosis in farm animals. Likewise, the various branches of veterinary medicine should be coordinated through research and practice to develop a comprehensive disease control program which will eventually reach the ultimate objective of eradication.

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Leptospirosis Zoonotic Reported

Investigation of a leptospirosis zoonosis in Iowa, by the Institute of Agricultural Medicine and the Iowa State Department of Health, traced 3 cases of leptospirosis in man to a swimming hole on a rural creek. Further investigation showed that 36 persons may have contracted leptospirosis in the same swimming hole and in other area creeks.

Cattle with access to streams were tested, and many were found serologically positive. Urine samples from 2 cows and 2 people yielded *Leptospira* organisms. Although typing of the organism was not completed at the time of this report, the character of the disease suggests *L. pomona* as the etiologic agent. —*Morbid. and Mortal. Rep., U.S. Dept. Health, Education, and Welfare*, 8, (Nov. 20, 1959): 2.

Epidemiology of Leptospirosis

in the United States and Canada

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OUR CURRENT KNOWLEDGE strongly indicates a marked increase in the prevalence of leptospirosis in both man and animals over the past decade. This increase may be accounted for, in part, by a greater awareness of the disease. It has been established that the epidemiology of the leptospires will follow a characteristic pattern based primarily upon the fact that they are zoonoses (diseases transmitted from animal to animal and from animal to man). Although this basic pattern has not changed, what are the conditions that have favored this increase in the spread of leptospirosis? Certain evidence is being accumulated which suggests the occurrence of changing conditions.

For many years, rats and dogs were considered to be the primary animal carriers of leptospires but, as the search for these organisms continues, the host range broadens not only among domestic animals but in a variety of wild mammals. Leptospirosis now constitutes a major problem in cattle and swine, and in some areas sheep, goats, and horses become infected. The rat is one of many rodent carriers including many species of mice, voles, and shrews.^{27,30,31} In addition bats, mongooses, bandicoots,² jackals,⁵⁴ hedgehogs, foxes, opossums, raccoons, skunks, and wildcats³⁴ have been found infected. In these host animals, leptospires become localized in the kidneys and may be found in the lumen of the convoluted tubules. After acute or even inapparent infection, these animal

carriers may become urinary shedders and serve as important foci of infection. Infections of man and other animals may result from direct or indirect contact with infected urine of these shedders.

Each leptospiral serotype usually is thought to have a primary animal host, but they may infect other animals, and a so-called "primary" host for 1 serotype may become infected with other serotypes or even harbor 2 types at one time.⁶⁰ A classic example of this is *Leptospira canicola* found principally in dogs; it has been isolated from cattle,^{52,55} swine,⁵⁶ jackals,⁵⁴ hedgehogs,⁵⁰ and serologic evidence suggests it may infect raccoons,⁴¹ while dogs have been found to harbor at least 9 other serotypes including *Leptospira pomona*.^{2,38}

In the United States, extensive studies on leptospirosis in domestic animals during the past decade have shown that it is a serious problem in cattle,⁶⁰ swine,¹² and dogs.⁶¹ However, until recently, investigations on potential wildlife reservoirs have been limited to rodents, particularly the Norway rat (*Rattus norvegicus*).^{16,30} In 1953 an epidemiological investigation of possible wildlife carriers of *L. pomona* in an enzootic area of bovine leptospirosis was conducted in Virginia.⁶² *Leptospira ballum* was recovered from 1 of 2 opossums (*Didelphis virginiana*) and from 9 of 27 house mice (*Mus musculus*). This represented the first reported isolation of *L. ballum* from the opossum. It was concluded that the failure to detect *L. pomona* in the rodent and other wildlife population in the area provided ancillary evidence that the natural hosts of this serotype were primarily in the livestock population.

Wildlife Reservoirs

Investigations concerning possible wildlife reservoirs of leptospires were initiated by the Communicable Disease Center in 1953 at Newton, Ga. During the first 2 years, these studies were confined to rats and mice.¹¹ *Leptospira ballum* was found frequently in house mice and occasionally

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in the roof rat (*Rattus rattus*), cotton rat (*Sigmodon hispidus*), and the old-field mouse (*Peromyscus polionotus*), but no other serotypes were encountered.

During a 6-month period ending in March, 1956,³⁴ 820 mammals including 14 species were trapped in different areas of 6 southwestern Georgia counties and suspensions of the kidneys were cultured. Leptospire isolates were obtained from 44 (5.4%). Of these, 16 proved to be *L. ballum*, 14 were *L. pomona*, 2 were *L. australis*, 2 were *L. grippityphosa*, 2 proved to be a new serotype of the *hyos* serogroup tenta-

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tively designated as *L. bakeri*²⁴ and 2 belonged to the *hebdomadis* serogroup. Two additional cultures were found to belong to the *hyos* serogroup, but are not identical with other recognized members of the group. Of the remaining 4, 2 were lost prior to serotyping and the identification of 2 has not been completed.

In this series, opossums, raccoons, striped skunks, foxes, and wildcats were found to harbor 1 or more of these leptospiral serotypes. These 5 species represented 79 per cent of the individuals examined. None of the infected animals showed any clinical signs of leptospirosis and no gross kidney lesions were observed. Only 4 of the 44 infected animals were determined to be less than 1 year old.

The highest incidence of infection appeared in striped skunks, with isolations from 18 (14%) of 132 animals. Eleven (61%) of these cultures proved to be *L. pomona*. On 1 farm in Peach County, Ga., 67 per cent of a herd of 96 beef cattle showed high agglutinin titers to *L. pomona* and 5 of the cows had aborted. Eighteen wild animals were trapped on this farm and *L. pomona* was isolated from 2 striped skunks. Cultures of *L. grippityphosa* were isolated from raccoons trapped within 50 miles of a Florida farm on which cattle showed antibodies to *L. grippityphosa* in 1952.²¹

These studies³³ have also yielded 7 isolations of *L. autumnalis* from raccoons trapped within a 2-mile area and 1 from an opossum. This represented the first reported animal host for this serotype in the United States, although it has been isolated from rodents in Japan⁵⁸ and dogs¹⁸ and bandicoots in Malaya. *Leptospira autumnalis* was first identified in this country by Gochenour *et al.*²⁶ in 1950 as the cause of epidemics of a febrile illness that occurred among troops at Fort Bragg, N. Car., during the summers of 1942, 1943, and 1944. The agent was isolated from 1 of the acutely ill patients and maintained by passage in guinea pigs and hamsters for 7 years.⁵¹ The possibility of an animal host of this serotype was not investigated during the Fort Bragg epidemics or after the causative agent was established. However, in October, 1957, a limited study²² yielded leptospire isolates from the kidneys of 10 (11.7%) of 84 wild animals trapped in this area. The infected animals included 5 raccoons, 3 gray foxes, and 2 red foxes. Three of the leptospiral strains isolated belonged to the *australis* (Ballico) serogroup, and 7 to the *grippityphosa* serogroup. While an animal host of *L. autumnalis* was not established during this limited study, leptospiral infection among the wild animal population appeared more prevalent than in the southwestern Georgia area.

Elsewhere in the United States only limited studies of the prevalence of leptospire isolates in small wild mammals have been conducted. Serological evidence was reported which suggested *L. canicola* infection in raccoons in New York.⁴¹ *Leptospira pomona* was isolated from opossums in Louisiana.⁴³ In a recent survey of wild animal sera in Ohio, by the Divisions of Wildlife and Animal Industry, Ohio Department of Agriculture,¹ leptospiral antibodies against *L. pomona* were detected in 43 (19%) of 224 samples from deer, 16 (22%) of 70 sera from raccoons, 14 (25%) of 55 sera from foxes, and 6 (54%) of 11 sera from skunks.

With the increase in the deer population observed in recent years in many areas of the United States and a lack of knowledge concerning prevalence of leptospirosis in deer, other states have directed their attention toward this possible wildlife reservoir. A serologic survey on 243 deer serum samples in Illinois¹⁷ revealed antibodies for *L. pomona* in 10.2 percent and for *L. grippityphosa* in 10.2 percent.

potyphosa in 9.8 per cent. In Minnesota *L. pomona* antibodies were found in 16 per cent of 187 samples,⁶¹ while a joint survey⁴⁷ in 11 southwestern states revealed leptospiral antibodies in only 1.73 per cent of 403 samples. In contrast, no antibodies for *L. pomona* were found in 628 samples from deer in Massachusetts.⁴²

Concern among cattlemen in Washington regarding the possibility of wild waterfowl serving as a source of leptospiral infection in cattle stimulated workers at the State College of Washington, College of Veterinary Medicine, to study the problem. *Leptospira pomona* antibodies were demonstrated in serums from chickens on a ranch where active bovine leptospirosis was diagnosed.²⁵ Antibodies for *L. pomona* developed when water contaminated with urine from a cow with leptospirosis was fed to chickens and ducks, but they were unable to isolate leptospires from the excreta of these birds.²⁸

Recently, the isolation of *L. bataviae* from the kidneys, liver, heart, and intestines of 5 species of wading birds captured in the rice fields of Italy has been reported.⁴ Leptospires were observed in cultures from 12 of 100 birds but contaminating organisms prevented isolation from 7 of these cultures. The birds are all migratory and leave Italy in the fall for southern and central Africa. If they can remain carriers of leptospires for long periods, they may serve not only as local sources of infection, but may also transmit leptospirosis between distant countries.

Although arthropod vectors have not been incriminated as yet in the transmission of leptospirosis in nature,^{13,46} several investigators have been able to infect ticks by allowing them to feed on infected guinea pigs or hamsters. These experimentally infected ticks transmitted the disease to normal animals. However, the isolation of *L. grippotyphosa* from the European tick *Dermacentor marginatus* S. has been reported from Russia.³² The ticks were from cattle where leptospirosis had occurred among the herd. More recently, *L. canicola* has been isolated from ticks of the Ixodid family, *Rhipicephalus sanguineus* collected from a hedgehog in Israel.⁵⁷ This provides further support to the suggestion that ticks may act as vectors of leptospires and that they should be considered in the infection chain until their importance is known.

Mode of Infection

During the acute phase of leptospirosis in lactating animals, leptospires may be shed in the milk, but no human cases have been attributed to drinking infected milk in the United States. This may be explained by the fact that whole milk is leptospirocidal and that organisms survive in it only a few hours.²⁹

The leptospires usually enter the body through the mucous membranes of the conjunctivae, nose, mouth, or abrasions on the skin. It is doubtful that these organisms penetrate the intact skin, and it is unlikely that the digestive tract is an important portal of entry since the pH of the stomach is usually such that the organisms may be quickly destroyed.

Environments that favor the survival of leptospires outside the body include moist soil, stagnant ponds, or slow-moving streams that are neutral or slightly alkaline, and a temperature of 22 C. or above. When these conditions exist in nature, leptospires may survive several weeks.^{14,48}

Under such environmental conditions, several epidemics have occurred in the United States following swimming in contaminated water. Since 1940, there have been 4 epidemics in Georgia,^{10,23,62} 1 in Alabama,⁴⁵ 1 in Wyoming,¹⁵ and 1 in Florida involving more than 160 individuals. All these water-borne leptospiral epidemics followed a similar pattern. They occurred in the late summer during drought periods; there was presumed contamination of a stagnant pond or slow-moving creek by urine from infected animals and transmission to persons by immersion in the contaminated water. The patients were young, primarily children 5 to 16 years old, but some young adults were also involved.

Occurrence in the United States

In recent years, a marked increase has been shown in the prevalence of leptospirosis in man in the United States. For example, 228 cases from 1905 to 1948 were reported in a summary.³⁵ In addition, 78 cases were reported from the Detroit area.³⁵ In contrast, a summary of information received by the Communicable Disease Center during a 6-year period (1953-1958) revealed a total of 533 cases in 43 states.

Opportunities for exposure are frequently encountered by veterinarians, animal husbandry men, swine herdsman, and workers in kennels, slaughterhouses, sewers, dairies, and poultry and fish houses.

During the past 5 years, information has been obtained at the Communicable Disease Center regarding 216 cases that occurred in 35 states and the District of Columbia.^{20,59} Information concerning the probable source was obtained on 146 of the 216 cases. Of these, 56 (38%) had had contact with infected cattle or swine either in abattoirs or on farms; 39 (26%) had been drinking, swimming, or accidentally immersed in presumably contaminated water; 21 (14%) had had contact with dogs in their homes or in veterinary hospitals; 19 (13%) were exposed to rats; 6 (4%) to wild animals; and 5 (3%) to other animals or possibly contaminated environments in their occupations. The fact that the probable source of more than one-third of these cases was found to be contact with infected cattle or swine may be attributed, in part, to the rapid spread of bovine leptospirosis in the United States.

Occurrence in Canada

Reported investigations on leptospirosis in both man and animals in Canada indicate that the disease is not as widespread as in the United States. Several cases of Weil's disease were reported between 1926 and 1950,^{6,7,40} but no other reports of human leptospiral infections appeared until 1955 when a case of "canicola fever" with meningitis was reported.⁴⁴ This patient had been bitten on the hand by a dog 1 week prior to onset. Later in 1955, a case of fever and meningitis was reported,⁹ in which serologic studies suggested infection with *L. sejroe*. Again, the source of infection was believed to be a dog. Both of these reports suggested that, in Canada, some cases of fever or meningitis of unknown origin may be caused by leptospires, and that, as laboratory diagnostic tests for leptospirosis are utilized in such conditions, these infections would probably be found more frequently.

Bovine leptospirosis was first reported in Canada in 1952⁴⁹ when leptospires were demonstrated in histopathologic sections from a calf. *Leptospira pomona* was not isolated from cattle until 1957;⁵ however,

a serologic survey in 1954³⁶ indicated that leptospiral infection was rather widely distributed in herds in Ontario and Quebec. A second survey in 1956⁸ revealed agglutinin titers against *L. pomona* and, to a lesser extent, against *L. sejroe* in 8 per cent of 2,695 cattle tested. These reactors were distributed throughout 60 per cent of the 113 herds represented.

No reports have been found regarding the prevalence of leptospirosis in dogs or in wild animals in Canada but, as it is known to occur in these animals in many of the border states in the United States, a similar distribution might be expected.

Discussion

Recent surveys to determine the prevalence of leptospires and leptospiral antibodies in wild animals in widely separated areas of the United States have established the existence of leptospirosis in a variety of species.¹⁹ Little is known of the nature of leptospiral infection in these wild animals, the duration of the carrier state, or their role in the possible transmission of the disease to man and to domestic animals. Obviously, further research is needed to clarify these facts.

Certain environmental factors such as temperature of 22 C., moist, neutral, or slightly alkaline soil, and water are known to favor the spread of leptospires. This lends credence to our belief that the apparent existence of an epizootic or panzootic period of the leptospires may be attributed largely to changing environmental conditions which are more favorable to the survival of the leptospires.

In Poland, it has been observed that leptospirosis belongs to the group of diseases in which development and epizootics depend largely on atmospheric conditions and frequency with which these conditions appear.³⁰ This observation explained the high frequency of swamp fever (*L. grippotyphosa* infection) in one local area in 1955 compared with its rare occurrence in 1956 and 1957 by climatic conditions prevailing at that period. Optimum meteorologic conditions for the survival of leptospires occurred in the spring and summer of 1955, but in 1956 and 1957 considerable variations were observed which apparently created unfavorable conditions for the leptospires.

Cultural and serologic studies on people and cattle in the United States have indicated that infection occurs in both with most of the serotypes that have been found in wild mammals. Further, it has been firmly established that transmission of leptospirosis is dependent upon an animal carrier that is shedding leptospires in the urine. Contamination of an environment suitable for survival of the organisms occurs by exposure to infected urine and by direct or indirect contact with these organisms by a susceptible person or animal.

What part do these wild animal reservoirs play in the chain of transmission? Are they an important link? As recently pointed out, the complexities of the problem in deer extend into conservation, agriculture, veterinary medicine, and public health.¹⁷ Hasty action for control, not based upon adequate research, may result in loss or injustice to one or more groups concerned. Certainly, the same may be said of other wild animal species until more information has been obtained.

The wide distribution of leptospires in the wild animal populations throughout the world presents a real problem in our efforts to control the disease in both man and domestic animals. Therefore, in spite of somewhat encouraging results with vaccines, leptospirosis may be expected to occur for many years to come.

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Leptospirosis

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R. W. H. GILLESPIE, PH.D.
L. M. RINGEN, PH.D.

ONE OF THE greatest handicaps to control of leptospirosis in domestic animals is lack of a practical diagnostic test which will recognize the animal capable of spreading infection. Bovine serums may react positively to present serologic tests for 3 to 6 years or more, while shedding of leptospire in urine is usually limited to about 3 months. A more suitable serologic test possibly is the hemolytic test based on the genus-specific erythrocyte-sensitizing substances.^{1,2} Bovine serums reacted positively to the hemolytic test for not more than 20 weeks in a group of cattle exposed to virulent *Leptospira pomona*, while leptospiruria was demonstrated for a maximum of 13 weeks after exposure.³ If further studies with field infections support the present findings, this test may be used as a more sensitive diagnostic aid to control leptospirosis.

The importance of water-borne leptospire has been emphasized by many early workers.⁴ In the United States, epidemics in man have been traced to swimming in water contaminated by livestock (*L. pomona* in Alabama,⁵ *Leptospira canicola* in Georgia⁶). *Leptospira pomona* may be present in surface waters contaminated by shedder cattle.⁷ Severe epizootics involving deaths and abortions (30%) as-

sociated with *L. pomona* infection have been observed in herds where a limited surface-water supply received heavy urine contamination.⁸ The alkaline surface waters of the Columbia Plateau favor survival of leptospire, and thus aid the spread of this disease. Consequently, a water source should be isolated to avoid contamination by livestock. The hazard of infection can be reduced by draining water pools in pastures, yards, and around watering troughs. A change of pasture may sometimes reduce exposure to contaminated surface waters.

Many practitioners report that leptospirosis "abortion storms" terminate within 7 to 10 days after vaccination. During the last 3 years, and particularly during the 1959 calving season, observations were made and reports received of abortions which have continued for 3, 4, or even 5 weeks after vaccination. One "abortion storm" involved 15 per cent of a 2,500 cow herd in which calf losses continued for 5 weeks after vaccination.⁹ Another herd involving 150 cows continued to abort for 30 days following vaccination.¹⁰ Since experimentally infected cows may abort 5 weeks after exposure, these observations were not unexpected. Apparently, the cattle which aborted after vaccination were already infected or became infected before the bacterin had stimulated sufficient protective immunity.

In an attempt to reduce this postvaccination period of noneffective immunity to a minimum, both experimental and field trials with a living egg-passaged (EP) (attenuated) strain of *L. pomona* have been initiated. We found that the agglutinins began to appear by the fourth day following vaccination with the living attenuated

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leptospirae, and within 6 days all vaccinated cattle had developed titers. In contrast, our bacterin studies indicated that at least 10 days were required before any vaccinated cattle developed a demonstrable titer.¹¹ No undesirable clinical effects have been observed in cows vaccinated (EP strain) during the last 3 months of gestation, either in experimental trials with limited numbers, or in field trials involving 30 to 150 cattle.

The use of attenuated living vaccines has been reported.^{11,12} Cattle vaccinated with attenuated living vaccine have been found resistant to challenge for at least 17 months,¹² though the Washington State workers consider their vaccinated cattle immune 3 years following vaccination.

In studies of an EP vaccine, a low level of shedding of attenuated organisms due to the vaccine has been observed which could not be detected except through demonstration of titers in urine-inoculated laboratory animals.¹¹ Shedding was detected for only a week or 2 in about one third of the yearling vaccinated cattle. On the other hand, a more delayed and protracted febrile response and shedding of organisms was observed in older cattle. Contact transmission was not observed. A mild nephritis was observed in some of these vaccinated cattle.

In 1 field trial involving a leptospirosis abortion storm, we were able to evaluate both the EP strain and the bacterin. The herd of 150 cows involved in this trial was allotted to separate groups of 75 each. One group received commercial bacterin and the other an EP strain of vaccine. Seventeen abortions from the 23rd to the 30th day after vaccination occurred in the group receiving bacterin. Six abortions from the 22nd to the 30th day after vaccination occurred in the group receiving the EP vaccine.

The judicious use of antibiotics may further reduce losses. For instance, feeding of antibiotics to leptospirosis-infected swine herds aided in reducing the abortion rate, leptospiuria, and baby pig mortality.^{13,14} and chlortetracycline in the feed prevented bovine leptospiuria.¹⁵

Although bacterins may give satisfactory results in many bovine leptospirosis epizootics, some complications associated with concomitant retention of fetal membranes are eliminated through feeding of chlortetracycline.¹⁰ Individual

treatment of aborting cows may be indicated in view of the mortality associated with retained fetal membranes. One report indicated that 5 of 9 cows died with 24 hours after aborting as a result of such complications.¹⁶ Clinical diagnosis of acute leptospirosis was confirmed by a diagnostic laboratory.

In another field observation⁹ involving 2,500 cows, 50 per cent of the 375 aborting *Leptospira*-infected cows developed complications associated with retained fetal membranes, resulting in a 4 per cent mortality.

Summary

With a bacterin, 10 days or more are required after vaccination to develop demonstrable serum antibodies, although abortions in vaccinated cows may continue for 3 to 5 weeks. Attempts to reduce such losses, which may approach 25 per cent or more, include: (1) vaccination at the earliest opportunity following the infection, together with administration of antibiotics parenterally, in feed, or in water; (2) parenteral antibiotic treatment of aborting cows to reduce death losses associated with retained fetal membranes; and (3) control of access to surface waters to reduce continued exposure of the herd.

Vaccination of cows with an egg-passaged attenuated living strain of *Leptospira pomona* during an epizootic has resulted in a 66 per cent improvement over results obtained with bacterin. This vaccine: (1) stimulated the formation of antibodies which appeared by the fourth day and persisted for at least 36 months, (2) produced minimal shedding in yearling cattle for 1 to 2 weeks which could not be determined by darkfield examination of the urine, (3) did not appear to interfere with normal parturition, and (4) was found nontransmissible from vaccinated to susceptible contact-control cattle.

Preliminary studies suggest that the hemolytic test may be an important aid in the control of bovine leptospirosis due to genus specificity of the antigen and to the relatively early disappearance of hemolytic antibodies after infection.

The effectiveness of antibiotic therapy appears to be reduced by any time lag between the onset of illness and the initiation of therapy.

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Nasal Discharge and Infectious Bovine Rhinotracheitis Virus

Eleven steers which had been maintained on full feed for 3 months were inoculated either intranasally or intramuscularly with a tissue culture-adapted strain of infectious bovine rhinotracheitis virus. The virus preparation had a titer of $10^{-7.0}$ and the dose was 4.0 ml. intranasally or 2.0 ml. intramuscularly.

Some or all of the symptoms associated with this disease, including fever, copious nasal discharge, difficulty in breathing, anorexia, and weight loss, developed in each animal. The 4 steers inoculated intranasally and 5 of the 7 steers inoculated intramuscularly developed typical copious nasal discharges.

Nasal swabs were collected on the first 3 days following appearance of nasal discharge and tested for virus. Virus was not found in any sample collected from the group inoculated intramuscularly but was found in all samples from the group inoculated intranasally.—[Albert L. Brown and C. B. Bjornson: *The Relationship of Nasal Discharge to Infection with Infectious Bovine Rhinotracheitis Virus*. *Am. J. Vet. Res.*, 20, (Nov., 1959): 985-988.]

Varicella (Chicken Pox)

in Three Young Anthropoid Apes

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VARICELLA is a relatively common condition in man, but a thorough search of the literature reveals that no cases have been reported in any other host through natural infection. This report is believed to be the first recorded incidence of spontaneous chicken pox (varicella) infection in any animal other than man.

In the Children's Zoo at the San Diego Zoological Gardens, 3 young anthropoid apes are exhibited in close contact with the public. These young apes participate in children's parties which are held in the Children's Zoo area and are handled and petted frequently. Outside the zoo, they have attended luncheons, dinners, theater openings, charity shows, and other functions.

Case Reports

Case 1.—"Tria," a female chimpanzee (*Pan troglodytes*), 3½ years old and weighing 30 lb., was examined at the Children's Zoo. She was discovered to have a macular rash, with 3-mm. lesions in each axilla, causing considerable pruritus. Her eyes, nose, and chest were clear and her appetite was good.

One week later, she was admitted to the zoo hospital with a papular rash on the chest, back, arms, and groin and with a few lesions on the chin. Rectal temperature was 103.9 F. (normal is 99.8 F.); slight diarrhea and inappetence were present. The buccal mucosa revealed numerous white vesicular lesions 1 mm. in diameter. Papules on the body were approximately 3

mm. in diameter. Tetracycline was administered orally. On the second day, several papules on the lower back and rump developed 1 mm. vesicles at their centers. The animal's appetite was good; rectal temperature had dropped to 100.4 F.; w.b.c. was 6,950; differential count was normal.

A pediatrician was called for consultation. Following his examination of the patient, the diagnosis of chicken pox was made. During the following 4 days, there was little change in general appearance; scabs were beginning to form over the lesions. Rectal temperature dropped to normal, appetite was good, and w.b.c. was 14,500.

During the following 4 days, scabs began to dry and fall off. On the nineteenth day after admittance to the zoo hospital, the animal was free of active lesions or scabs and was released to the Children's Zoo.

Case 2.—"Noell," a female orangutan (*Pongo pygmaeus*), 2½ years old, entered the hospital one week after "Tria," the chimpanzee, was admitted. She was also a victim of constant pruritus. No abnormalities were found in the eyes, nose, or chest on physical examination, but she was lethargic. No skin lesions were apparent until the third day when several typical white vesicles, 1 mm. in diameter, appeared on the buccal mucosa and one 2-mm. papule was found in the left axilla.

On the fourth day, she was examined by the pediatrician, who diagnosed chicken pox. On the seventh day, she developed a cough and sneezing; there were several lesions (2-mm. papules with small central vesicles) on both arms and axilla and she scratched her rump and thighs. On the eighth day, she was lethargic; axillary and inguinal lymph glands were enlarged; and the papules developed into vesicles.

During the following 4 days, scabs

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The author thanks Lawrence G. Reck, M.D., pediatrician, San Diego, Calif., for his valuable aid in consultation on the cases herein described.

formed over the lesions and sloughed off. She was released the next day with Tria.

Case 3.—"Scoop," a lowland gorilla (*Gorilla g. gorilla*), male, approximately 20 months old, had been in the Children's Zoo only 2 months. On the eleventh day after Tria had entered the hospital, he was observed scratching his right axilla. Examination of the area revealed several 2-mm. papules. On the following day, these lesions had disappeared and he was no longer scratching. He was not admitted to the hospital.

Discussion

The disease described above occurred during the midsummer, at which time there was a high incidence of chicken pox in children in San Diego County. Although no laboratory procedures were utilized to confirm the diagnosis, it is believed that the signs, course, and lesions exhibited by these animals were typical of human varicella.⁴

The development of intranuclear inclusion bodies in the testes of monkeys which were given intratesticular inoculations of material from human varicella lesions has been reported.³

Experimentally produced varicella in monkeys following inoculation by intranasal, intraperitoneal, and subcutaneous routes has been reported.² An incubation period of 5 days was followed by the development of typical lesions on the face, trunk, and in some cases in the mouth, pharynx, and on the extremities.

A search of medical and biological references, however, has failed to disclose any previous report of natural infection of subhuman primates with the varicella virus.

The mode of exhibition of young great apes in close contact with human beings, as practiced at the San Diego Children's Zoo, is unique, and it may be that this situation afforded a better opportunity for exposure of these animals to an infectious dose of virus particles. Most zoos isolate their great apes from the public by exhibiting them behind glass or at some distance from the viewers, in which case exposure to human infectious agents is limited. The chimpanzee exhibited a typical course of the disease, while the orangutan and gorilla were less severely affected. This difference would presumably be due

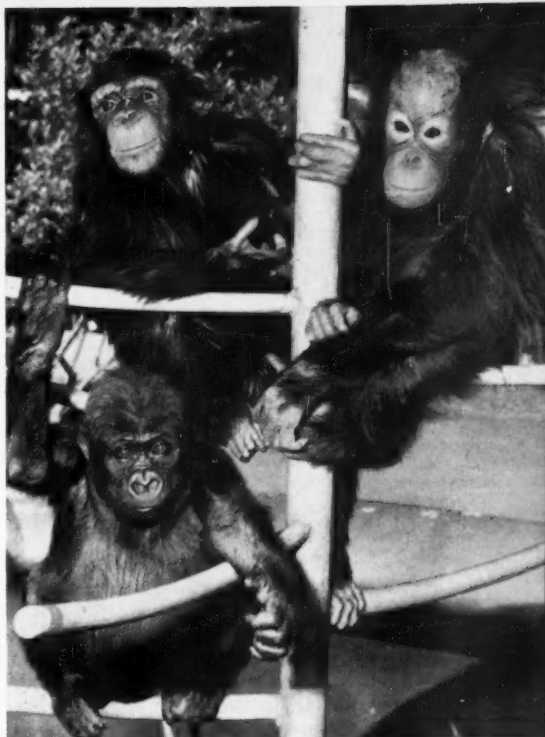


Fig. 1 — These three young anthropoid apes are believed to be the first nonhuman animals to become naturally infected with chicken pox.

either to difference in individuals or species differences in susceptibility, or to exposure to a small amount of virus.

Summary

Chicken pox (varicella) developed in 3 young anthropoid apes (a chimpanzee, an orangutan, and a gorilla). Signs, course, and lesions were typical of the average case in man. This is believed to be the first report of natural infection of any animal other than man with this disease agent.

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Canine Scapula

— A Case Report

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L. E. EVANS, D.V.M., M.S.
J. M. DAVIS, D.V.M.

AFTER BEING HIT by a car, a male Pointer dog, 4 years old, was admitted to a private practitioner's (J.M.D.) hospital for observation and treatment. The dog was unable to extend the right thoracic limb. There was an area of subcutaneous emphysema posterior to the right shoulder. Radiographs showed no fractures of ribs or limb. Routine treatment for shock was initiated, and the right thoracic limb was snugly taped to the thorax in a flexed position. Later examination of the patient revealed that a peculiar shoulder injury was present, and the animal was referred to the Auburn University small animal medical center for further observation and possible treatment.

Upon admission, the patient showed signs of weakness of shoulder girdle muscles. When he placed weight on the right thoracic limb, the proximal border of the right scapula was forced upward 3 or 4 inches above the level of the back. A soft, spongy mass of tissue was palpated between the skin and spine of the right scapula. The nerve supply to the limb was apparently not impaired because reflexes in the limb were normal (fig. 1).

Diagnosis and Discussion

A tentative diagnosis of rupture of the right serratus ventralis and trapezius muscles was established, assuming that the

ruptured serratus muscle allowed the abnormal upward deviation of the scapula. This diagnosis was based on the fact that the serratus ventralis muscle is the only muscle of the shoulder girdle that limits the upward movement of the entire scapula. Limitation of the upward deviation of the scapula by this muscle is due to its ventral attachment to the cervical vertebra and sternal ends of the ribs and its dorsal attachment to the proximal border of the scapula (fig. 3). The normal action of the serratus ventralis is to raise the thorax.

Rupture of the trapezius muscle appeared to have no bearing on the attitude of the limb since the cervical and thoracic parts of this muscle act together in elevating the scapula. Since the abnormal deviation of the shoulder would have impaired permanently the dog's hunting ability, surgical repair was deemed advisable.

Procedure

Surgical repair involved 2 operations. In the initial operation, an incision was made through the skin, fascia, and rhomboideus muscle parallel to the proximal border of the scapula. The trapezius muscle had been ruptured at its origin. The medial surface of the serratus ventralis muscle was exposed, revealing ruptures at both its ventral and dorsal attachments.

Musculature and fibrosis present on the medial surface of the scapula were secured to the thoracic wall by several interrupted $\frac{3}{8}$ -inch umbilical tape sutures. Musculature, fascia, and skin were sutured

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Fig. 1—Photograph of dog (left side) showing right scapula displaced upward 3 to 4 inches above left scapula.



Fig. 2—Right side view shows normal position of right scapula following surgical repair.

routinely. The limb then was placed in a flexed position and immobilized by fixing it to the thoracic wall by means of plaster cast bandage material.

Two weeks later, the cast was removed from the patient. Scapular movement was much more stable; however, upward dislocation was still 1.0 to 1.5 inches higher than the opposite scapula. Therefore, a second surgical procedure was initiated.

The incision and approach was identical to that used in the first operation. Then 2 holes were drilled lateromedially through posterior portions of the scapula. Stainless steel wire was placed through holes around the seventh rib and tied. The areas of the incision were debrided and sutured again with umbilical tape ($\frac{3}{8}$ in.). The limb was taped in flexion to the thoracic wall for 2 weeks. At the end of this time, the scapula was in a normal position, and the dog could walk normally (fig. 2). The dog was discharged, and the owner was advised to gradually condition him for hunting.

Six weeks later, the owner reported that the dog had shown good recovery and use of the limb and was hunting 3 to 4 hours daily.

Summary

A case of luxation of the scapula in a male Pointer dog resulted from multiple muscular ruptures and tearing of attachments of the serratus ventralis and trapezius muscles. Surgical repair was effected in 2 stages, and recovery was complete.



Fig. 3—Lateral view of dissected specimen shows musculature with right limb reflected upward. (1) serratus ventralis, thoracic part; (2) teres major; (3) subscapularis; (4) serratus ventralis, cervical part; (5) superficial pectoral; (6) deep pectoral; and (7) scalenus muscles.

Barbiturate Antagonists

L. C. WEAVER, PH.D.
C. A. BUNDE, PH.D., M. D.

ALTHOUGH average doses for the different barbiturates have been worked out properly, it is obvious from experience that the administration of the barbiturate becomes an individual problem with each animal. It is not uncommon to see shock and respiratory arrest occurring in animals in which less than the full calculated dose has been employed. Preanesthetic drugs may produce a variable degree of respiratory depression which is additive to the depression accompanying the barbiturate. Daniel *et al.*² have recently shown that barbiturates produce more depression of the cardiovascular system even under controlled anesthetic conditions than was previously recognized. Thus, the cardiovascular system as well as the respiratory system should be considered when attempts are made to antagonize barbiturates. If lethal doses of barbiturates (*i.e.*, doses producing respiratory arrest) directly depress cardiac function, artificial respiration and cardiac stimulants would be indicated. Sympathomimetic amines have been considered clinically to combat hypotension in severe barbiturate intoxication in the expectation that they might overcome central depression (*eg.*, amphetamine) or peripheral vasodilation (*eg.*, phenylephrine).³ The study of Daniel *et al.*² suggests that the efficacy of such agents may well be the result of cardiac stimulation.

Methods

Seventeen adult mongrel dogs, unselected as to sex, were used for these experiments. The analeptics used were 3-bromo-sulfolane (3-bromo-tetrahydrothiophene-

1, 1-dioxide^{1,4}; pentylenetetrazol⁵; and, because of its desirable duration of action, the sympathomimetic, phenylephrine hydrochloride.^{**} The anesthetic agent was a pentobarbital sodium solution.[†]

Results and Discussion

Pentobarbital was administered by the intraperitoneal (*i.p.*) route until respiratory arrest and cardiac failure ensued (1 to 3 hr.). This route was used because the slow administration of the barbiturate produces a reliable, deep anesthesia that eliminates the possibility of spontaneous recovery which could occur following respiratory arrest produced by a rapid intravenous administration of the barbiturate. Indeed, the intoxication produced by *i.p.* administration should be the more difficult to antagonize. Typical results showing spontaneous recovery of respiration and systemic blood pressure due to bromosulfolane-phenylephrine administration are presented (fig. 1A) following a brief interval of artificial respiration (fig. 1B).

These results suggest that the phenylephrine at this dose may well produce some cardiac stimulant effects. These effects are most likely reflexly produced although the possibility of direct effect was not eliminated. Thus, the phenylephrine improves a depressed cardiovascular system which, in turn, is optimal for transporting the analeptic to the central nervous system where it produces its respiratory stimulant effect.

From a study² of barbiturates in dogs, it was concluded that electrocardiograms are inadequate for estimating the degree

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*Metrazol, Bilhuber-Knoll Corp., Orange, N.J.

**Neo-Synephrine, Winthrop-Stearns, Inc., New York.

†Somnopentyl, Pitman-Moore Co., Indianapolis, Ind.

**BROMOSULFOLANE - PHENYLEPHRINE ANTAGONISM OF PENTOBARBITAL
INTOXICATION IN DOGS**

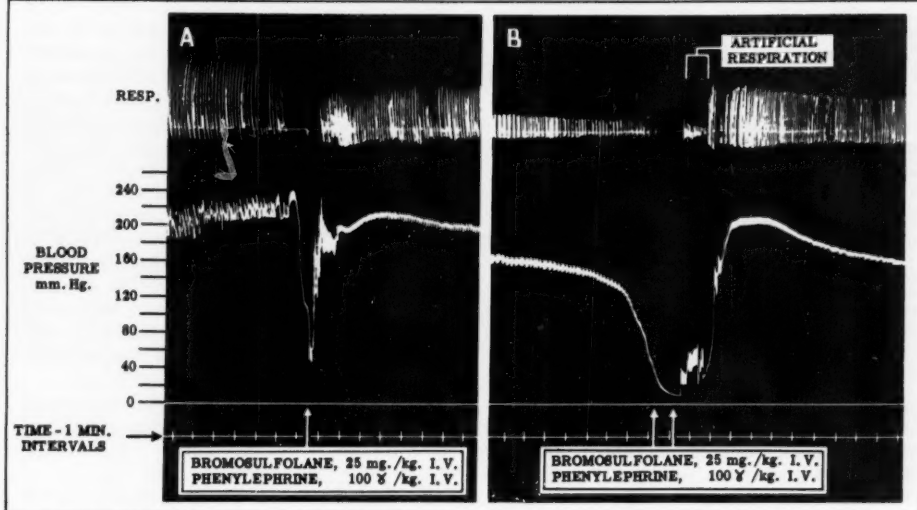


Fig. 1 — Tracings illustrating bromosulfolane-phenylephrine antagonism of pentobarbital intoxication. (A) Dog had received 87 mg./kg. of pentobarbital sodium over a 3-hour period. (B) Dog had received 67 mg./kg. of pentobarbital sodium over a 6-hour period. This dog received a similar dose of the combination 3 hours prior to this dose.

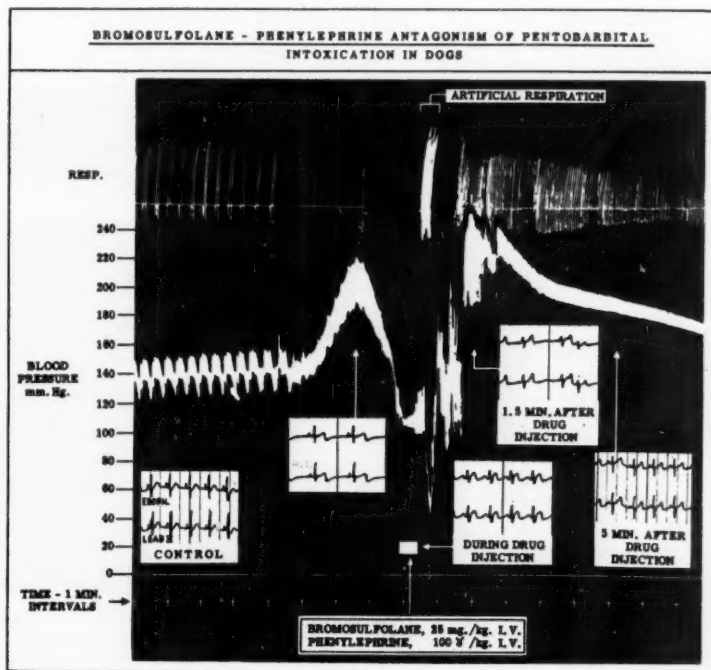


Fig. 2 — Tracing illustrating bromosulfolane-phenylephrine antagonism of pentobarbital intoxication. Dog has received 112 mg./kg. of pentobarbital sodium over a 3½-hour period. One hour prior, this dog had received pentylene-tetrazol plus phenylephrine.

of myocardial depression produced by barbiturates. Their usual records show little alteration other than terminal bradycardia and a moderate increase in PR interval. These results are confirmed in our studies. Typical electrocardiograms (lead II and esophageal lead, Gilson Electroencephalograph) before and after the bromosulfolane-phenylephrine combination in the pentobarbital-intoxicated dog are shown (fig. 2). It can be seen in the electrocardiogram taken during the blood pressure rise following respiratory arrest that the record shows a pronounced bradycardia, a slight prolongation of the PR interval, and T-wave alteration. Five minutes after drug administration, the record was similar to control.

Dogs deeply anesthetized responded well to i.p. administration of bromosulfolane-phenylephrine (fig. 3). There was a rapid onset of respiratory effects which lasted 30 minutes to 2 hours; there was no appreciable effect on systemic blood pressure.

The use of analeptic-sympathomimetic combinations was not without side effects.

When the bromosulfolane-phenylephrine combination was administered rapidly (1 minute or less) intravenously (i.v.) in an animal not in deep depression, or if the dose was too large, convulsions occurred. These were due to the analeptic and were moderately severe but usually had a duration of less than 1 minute. It is not necessary to use methods to abort these convulsions. Vomiting, which frequently follows convulsions produced by analeptics, created more of a problem since this occurred during or near the end of convulsions at a time when respiration was highly stimulated. Vomiting should seldom be a problem since it was not observed except in lightly anesthetized animals following immediate (10 sec. or less), i.v. administration.

Similar effects to all those mentioned above for the bromosulfolane-phenylephrine combination were also noted for a combination of pentylenetetrazol, 25 mg./kg. of body weight, plus phenylephrine hydrochloride, 100 γ /kg.

These data suggest that an analeptic-

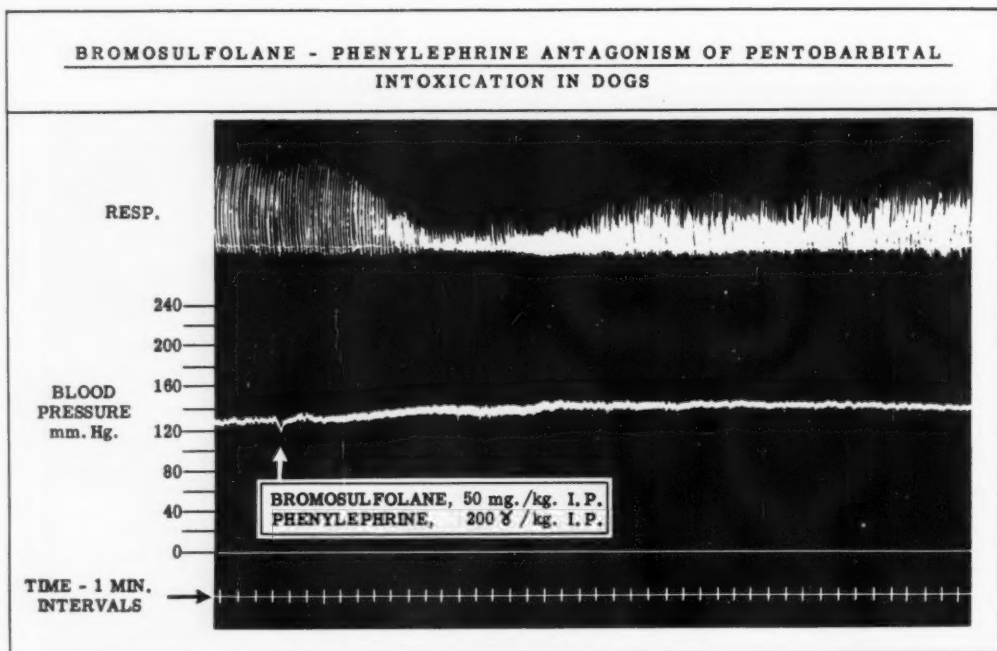


Fig. 3 — Tracing illustrating bromosulfolane-phenylephrine antagonism of pentobarbital intoxication. Dog had received 52 mg./kg. of pentobarbital sodium over a 1½-hour period.

sympathomimetic combination could be expected to lighten anesthesia, shorten anesthetic time, restore reflex activity and decrease the need for medical attention. However, it should be kept in mind that repeated injections may be necessary following intoxication due to the longer acting barbiturates. The side effects would be of little concern after experience is gained.

Summary

Dogs were given repeated doses of pentobarbital sodium by the intraperitoneal route until respiratory arrest and cardiac failure ensued. Attempts to antagonize this barbiturate-depressed state were successful with a combination of an analeptic (bromosulfolane or pentylenetetrazol) and a sympathomimetic (phenylephrine). It is suggested that such combinations may

prove useful in the clinic since they antagonize barbiturate effects on the cardiovascular system as well as the respiratory system.

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Bat Rabies Widespread

The following items are from the weekly *Morbidity and Mortality Reports* of the U.S. Department of Health, Education, and Welfare.

In Wisconsin, a 44-year-old farmer was bitten on the ear lobe by a bat on Aug. 8, 1959. The bat was killed by the victim, disposed of, and later eaten by the family cat. Approximately 3 weeks later, the farmer developed an illness which followed the pattern of rabies and resulted in death September 4. Mouse-inoculation tests proved the presence of rabies virus. The cats and dogs on the farm were destroyed without brain specimens being submitted for examination.—8, (*Oct. 2, 1959*): 2.

The first suspected case of rabies in a bat in Maryland was found in Baltimore County during late summer, 1959. The bat was killed by a group of children. Mouse-inoculation tests were performed and the first mouse deaths occurred 21 days after inoculation. Inclusion bodies were found in the bat's brain but were not entirely typical of Negri bodies.—8, (*Nov. 14, 1959*): 2.

The first known instance of bat rabies in Connecticut was confirmed by examination of the brain of a bat submitted to a laboratory by residents of Westport in October, 1959. The bat was picked up from the ground where it had fallen.

A dead bat was found in a residential section of Henrico County in Virginia in October, 1959. It was proved by mouse-inoculation tests to have been rabid. Direct examination of the bat's brain revealed findings suspicious but not clearly diagnostic of rabies.—8, (*Oct. 30, 1959*): 2.

The first case of bat rabies in the state of West Virginia was confirmed in August, 1959, by an animal-inoculation test. A 7-year-old boy had been bitten when he picked up a bat after it had fallen into a swimming pool. The bat was confined and died the following day. The boy was given antirabies treatment.

Laboratory tests in Nebraska in August proved that 2 bats found in different Nebraska counties were rabid. One was a

little brown bat which was found unable to fly and saliva was seen exuding from its mouth. Microscopic examination did not reveal Negri bodies, but mouse-inoculation tests were positive for rabies. A red bat found in a yard by a 9-year-old girl

was shown to be rabid on the basis of presence of Negri bodies and mouse-inoculation tests. The girl was bitten attempting to handle the bat which was finally captured by throwing a coat over it.—8, (Oct. 16, 1959): 2.

Immune Response of Dogs to Canine Distemper and Measles Viruses

Distemper-free pups were vaccinated with live canine distemper virus or inactivated or live measles virus and subsequently challenged with virulent canine distemper virus. The Lederle, Onderstepoort, Snyder Hill, and Wisconsin strains of canine distemper virus and the Edmonston strain of measles virus were used in this study.

Distemper-neutralizing and measles complement-fixing (CF) antibody titers of pre- and postvaccination and postchallenge serums were determined to ascertain whether dogs vaccinated (1) with live canine distemper virus develop antibodies to measles, (2) with inactivated measles develop measles or distemper antibodies, and (3) with live measles elicit a clinical and immunologic response. Dogs vaccinated with live egg-adapted distemper vaccine develop low titers of measles CF antibodies.

Following exposure to virulent distemper virus, an elevation of both distemper and measles antibody resulted.

Dogs inoculated with killed measles vaccine, however, failed to develop measles or distemper antibodies, but reacted like hyperimmune animals when challenged with distemper. Dogs infected with measles uniformly developed subclinical infections with the subsequent appearance of both measles and distemper antibodies. Measles-convalescent dogs were found to be resistant to virulent distemper challenge.

The findings substantiate the existence of an antigenic relationship between strains of canine distemper and measles and also suggest an antigenic mosaic or gradient with certain distemper strains being more closely related to measles virus than others.—[Joel Warren, Marvin K. Nadel, Evan Slater, and Stephen J. Milian: *The Canine Distemper-Measles Complex. I. Immune Response of Dogs to Canine Distemper and Measles Viruses. Am. J. Vet. Res.*, 21, (Jan., 1960): 111-119.]

Effect of Hyaluronidase on Rabies Virus

Addition of hyaluronidase to suspensions of virulent rabies virus given intramuscularly to dogs, in Germany, had no noticeable influence on the course of the disease.

When hyaluronidase was added to suspensions of fixed virus, intramuscular injections into the neck muscles of dogs resulted in clinical rabies. In subpassages, no hyaluronidase was needed to produce similar signs.

When hyaluronidase was added to chicken embryo-adapted Flury strain rabies virus, intramuscular injections into the neck muscles caused a temporary disease resembling clinical rabies in 3 dogs. From the brain of another dog which became totally paralyzed and was euthanatized, rabies virus was recovered.—H. Bindrich and A. F. Olechnowicz in *Archiv für Exptl. Vet.-med.*, 13, (1959): 523.

Heart and Great Vessels

in Healthy Living Dogs

Robert L. HAMLIN, D.V.M.

THE PURPOSE of this report is to describe radiographic relationships of the cardiac chambers and great vessels to each other and to the thorax of normal, living dogs. This information should provide better understanding of transmission of cardiac sounds,⁴ genesis of the electrocardiogram,³ interpretation of thoracic radiographs, and of other pertinent clinical and research procedures.

Topographic descriptions of cardiac anatomy^{1,5,6} have been obtained for the most part from dissection of formalized cadavers distorted by the fixative and placed in nonstandardized, nonvital postures. Using the angiocardiographic method of injecting radiopaque material selectively into various cardiac chambers and great vessels of the living dog,² and taking lateral and dorsoventral radiographs, a more vital and clinically applicable description of topographic cardiac anatomy may be obtained.

Materials and Methods

Groups of animals.—The 26 dogs used in this study were allotted to 2 groups. Group 1 consisted of 21 healthy, mature, mongrel dogs of equal sex distribution. Group 2 consisted of 5 dogs with clinical cases of mitral insufficiency, which were obtained from the Ohio State University veterinary clinic.

Radiopaque Material.—Radiopaque contrast material was a clear, moderately viscous, sterile, aqueous solution supplied

in 50-cc. vials.* Constituents were 85 per cent methylglucamine diacetylaminotriiodobenzoate, equivalent to 64.5 per cent base, and 3,5-diacetyl-amino-2,4,6-triiodobenzoic acid. The base contained 62 per cent iodine, and the solution approximately 40 per cent firmly bound iodine. Sodium citrate was added as a buffer, and disodium ethylenediamine tetra-acetate dihydrate as a sequestering agent.

Radiological Technique.—All radiographs were taken at 0.1 second exposure with 40-inch anode-film distance. Sante's rule was used for calculating the necessary peak kilovoltage. Milliampereage was adjusted to obtain 10 ma./sec. Intensifying screens were used in all cassettes.

All dogs were anesthetized with pentobarbital sodium, 35 mg./kg. of body weight. To obtain a lateral thoracic radiograph, the dog was placed with its right side on the cassette; and to obtain a dorsoventral radiograph, the sternum was placed on the cassette. The dogs were carefully positioned to assure precise superimposition of sternum and vertebrae on the dorsoventral radiographs.

Sequence of radiographs for group 1 dogs:

Number	View	Chamber injected
1	Lateral	None
2	Dorsoventral	None
3	Lateral	Right ventricle
4	Lateral	Left ventricle
5	Dorsoventral	Right ventricle
6	Dorsoventral	Left ventricle

All injections consisted of 15 cc. of the radiopaque material and were given through a 2-inch, 15-gauge hypodermic needle connected to a 20-cc. syringe by a

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*This product, Cardiograffin, was supplied by E. R. Squibb and Sons, New York, N. Y.

3-way stopcock and occasionally by polyethylene tubing. Animals in group 2 were injected only into the left ventricle and only in the lateral recumbent position.

Cardiac Punctures.—Direct cardiac puncture into the right ventricle was accomplished by extending the left thoracic limb and inserting the needle, directed caudad at approximately a 30-degree angle with the median sagittal plane, through the left second intercostal space immediately dorsal to the left sternal border. The needle was advanced until cardiac pulsa-

tion was felt, then quickly thrust 2 cm. deeper to enter the right ventricular lumen. The 15 cc. of radiopaque material was injected and, simultaneously with the completion of injection, the radiograph was taken. The needle was then quickly withdrawn.

Left ventricular puncture was accomplished by inserting the needle through the left fifth intercostal space and directing it medially and approximately 15 degrees cephalad from the point approximately one third of the ventrodorsal distance from the

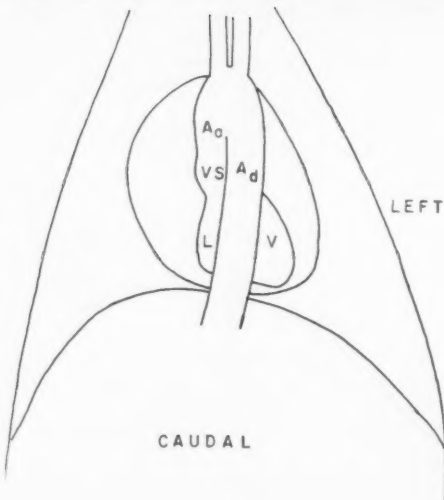
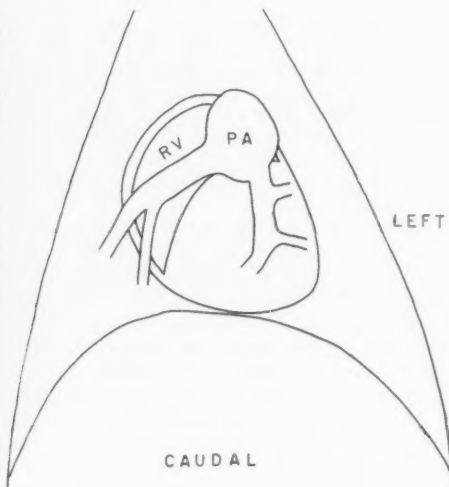


Fig. 1—Dorsoventral radiograph of normal canine thorax with schematic diagrams showing contribution of: right ventricle (RV), pulmonary artery (PA), left ventricle (LV), ascending aorta (Aa), descending aorta (Ad), and Valsalva sinuses.

sternum. Entrance into the left ventricular lumen, injection of the radiopaque medium, and exposure of the film were achieved as with right ventricular puncture.

When direct cardiac puncture was performed on animals which were to survive, the necessary areas were surgically prepared and aseptic precautions were observed.

Lead aVF electrocardiograms were taken on animals before, during, and after angiocardiology.

Results

Radiopaque Material.—Diatrizoate methylglucamine is an effective and safe radiopaque material. It has the advantage of high concentration, which allows for maximal contrast using a small volume; at the same time, it remains in solution at room temperature. Heating or agitation is seldom necessary before withdrawal from the vial and injection.

As high as 15 cc./kg. of body weight, injected intravenously over a 1-hour period,

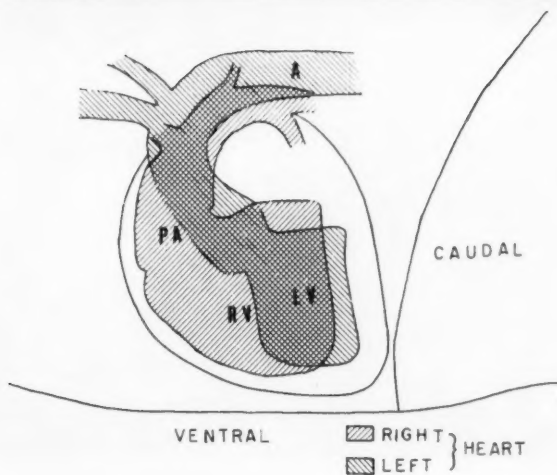
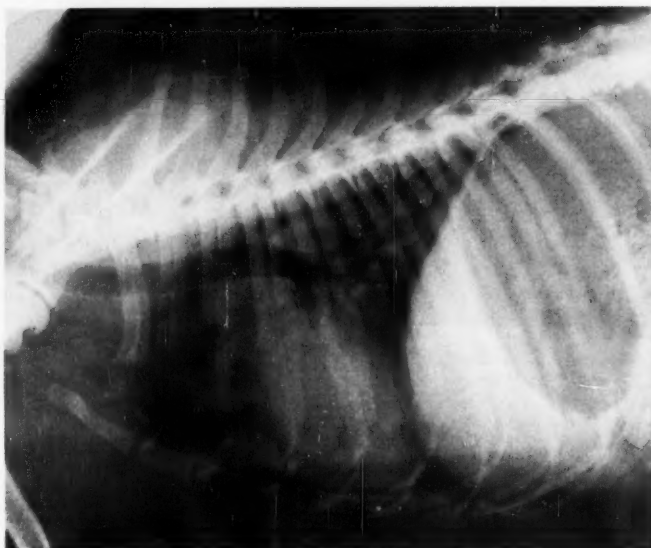


Fig. 2—Lateral radiograph of normal canine thorax, with schematic diagram showing contribution of left ventricle (LV), aorta (A), right ventricle (RV), and pulmonary artery (PA).

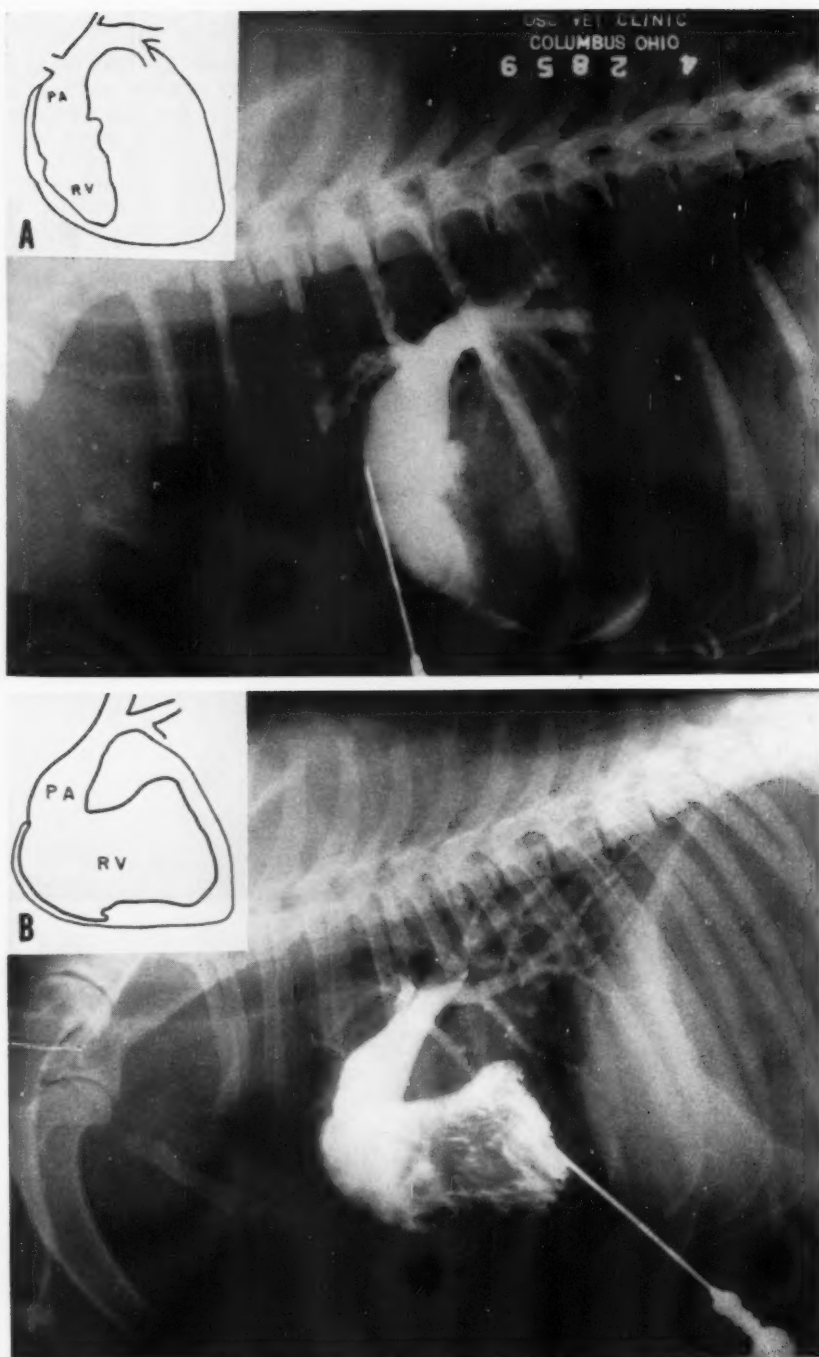


Figure 3

resulted in no abnormal blood pressure, heart rate, or electrocardiogram response. In more than 50 dogs, calves, and sheep injected with this material, in this and previous studies, no fatalities were observed. Occasionally, following cardiac puncture and rapid intracardiac injection, unifocal ventricular premature beats were observed briefly. In 25 per cent of the lightly anesthetized animals, 3 to 5 seconds following injections of radiopaque solution into the left ventricle, transient (15 sec.) excitement and hyperventilation were observed. Animals receiving the radiopaque injections inadvertently either perivascularly, intrapericardially, or intrapleurally showed no apparent discomfort or other adverse effect during 2 weeks' surveillance.

Dorsoventral Radiograph.—The cardiac silhouette in the dorsoventral radiograph (fig. 1) of the normal dogs in this study shows an elliptical area located in the mid-thorax, and extending from the third to the eighth rib, with slightly over one half of the mass to the left of the midline. The left caudal border is usually slightly closer to the left ribs than is the right cephalic border to the right ribs; however, animal-to-animal variation prohibits a conclusive statement for all normal dogs. The long axis of the heart forms an angle of approximately 30 degrees with the median sagittal plane.

The silhouette of the heart in the dorsoventral radiograph has a rounded right cephalic and right border, a near 90-degree angle at the left cephalic border, and a slightly elliptical form to the left caudal and caudal border. The right ventricle comprises the cephalic and right borders of the shadow. The left cephalic 90-degree angulation is formed by the pulmonary conus, and the left caudal and caudal border is produced by the left ventricle.

Occasionally a normal cephalic protrusion

in the median sagittal plane is formed by the aortic arch and the 2 cephalad coursing great arteries, brachiocephalic and left brachial. In properly exposed radiographs, an area of greater density may be observed slightly to the left of the midline and extending from the most cephalic cardiac extremity to and through the hepatic density. This increased density is due to the aorta as it leaves the center of the cardiac silhouette, proceeds cephalad, and then loops back upon itself, offset slightly to the left of the midline as it courses caudally into the abdomen.

The atria probably contribute little to the cardiac silhouette in the dorsoventral radiograph; however, the posterior vena cava along with the pericardio-diaphragmatic ligament and the pericardial fat may be visualized as a fan-shaped density with its apex at the caudal cardiac border on the midline and the base fusing into the hepatic density.

Lateral Radiograph.—The cardiac silhouette in the lateral radiograph (fig. 2) of the normal dogs in this study is a tear-shaped area located, for the most part, in the ventral portion of the thorax with the apex on the ventral wall of the thoracic cage between the diaphragm and the sternum. From the apex, the density fans out dorsad and cephalad forming a cone with a large base extending approximately two thirds of the ventrodorsal distance from the sternum to the extremity of the dorsal spinous process of the seventh thoracic vertebra. The longitudinal axis of the cone forms an angle with the ventral plane, of approximately 45 degrees. As in the dorsoventral radiograph, the heart shadow extends from the third to the eighth rib.

The cardiac silhouette in the lateral radiograph is rounded on the dorsal, ventrocephalic, and cephalic borders. It is almost flat on the caudal border and pointed at the ventrocaudal border. Occasionally, a ventral border indentation may be visualized where the right and left ventricles fuse into the interventricular septum. The cephalic border is occupied by the right ventricular free-wall; the caudal border by the left ventricular free-wall. Similarly, the cephalic and caudal ventral borders are formed respectively by the right and left ventricular apices. The dorsal-cephalic border is formed by the right atrium, pulmonary artery, aorta and anterior vena cava; and the dorsocaudal border is formed by

Fig. 3—Left lateral radiographs and schematic diagrams of normal canine thorax with right ventricle (RV) and pulmonary artery (PA) opacified. Notice the incomplete opacification of the narrow right ventricular extension superimposed upon the left ventricular shadow (fig. 3b). In this instance, right ventricular puncture was performed by inserting the needle through the left eighth intercostal space and entering the chamber from its most caudal aspect (the inflow tract).

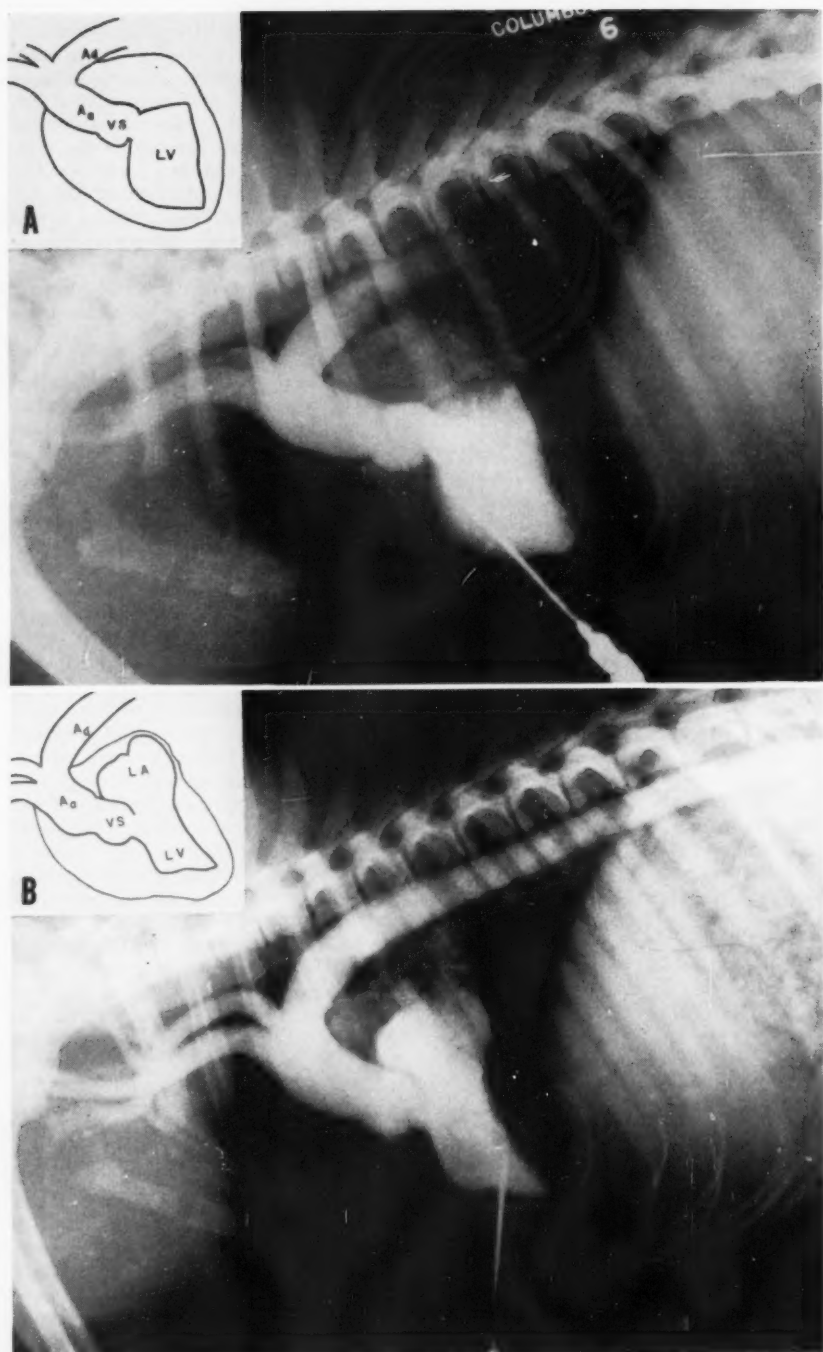


Figure 4

Fig. 4—Left lateral radiographs and schematic diagrams of normal canine thoraces with left ventricle (LV), Valsalvasinuses (VS), ascending aorta (Aa), and descending aorta (Ad) opacified. The left atrium (LA) is opacified as the radiopaque material regurgitated through an incompetent mitral valve (fig. 4b).

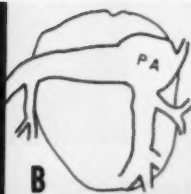
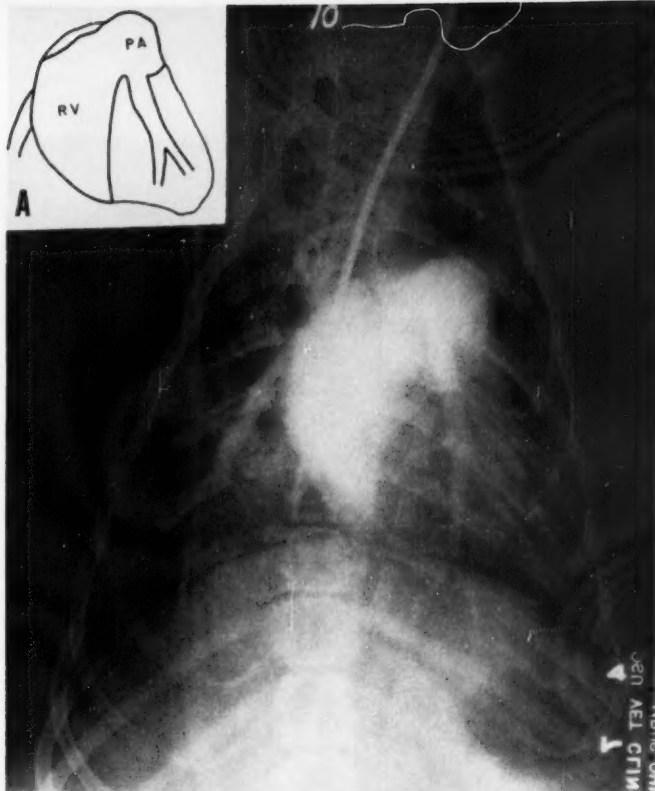
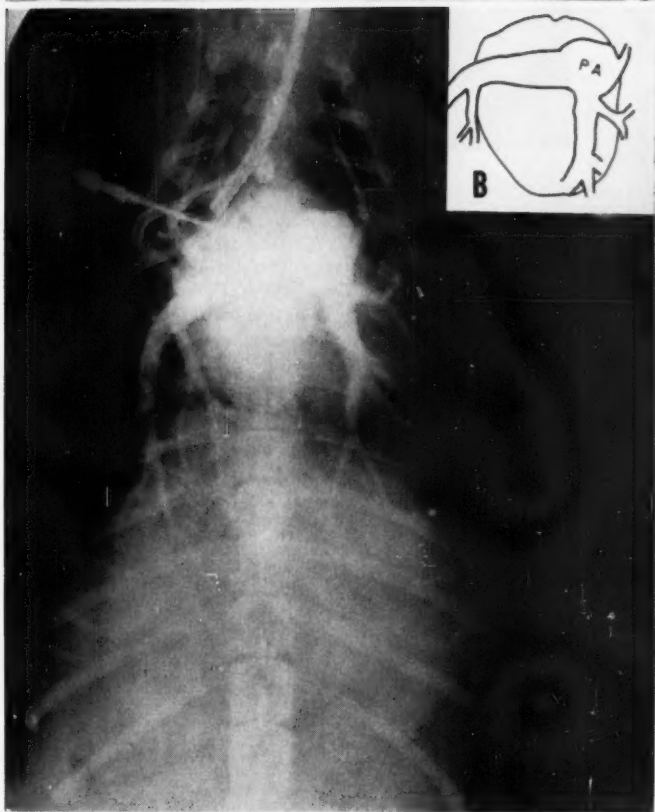


Fig. 5—Dorsoventral radiographs and schematic diagrams of normal canine thoraces with right ventricle (RV) and pulmonary artery (PA) opacified. Only the pulmonary artery is opaque (fig. 5b). A cardiac catheter located in the right ventricular lumen may be observed (fig. 5a).



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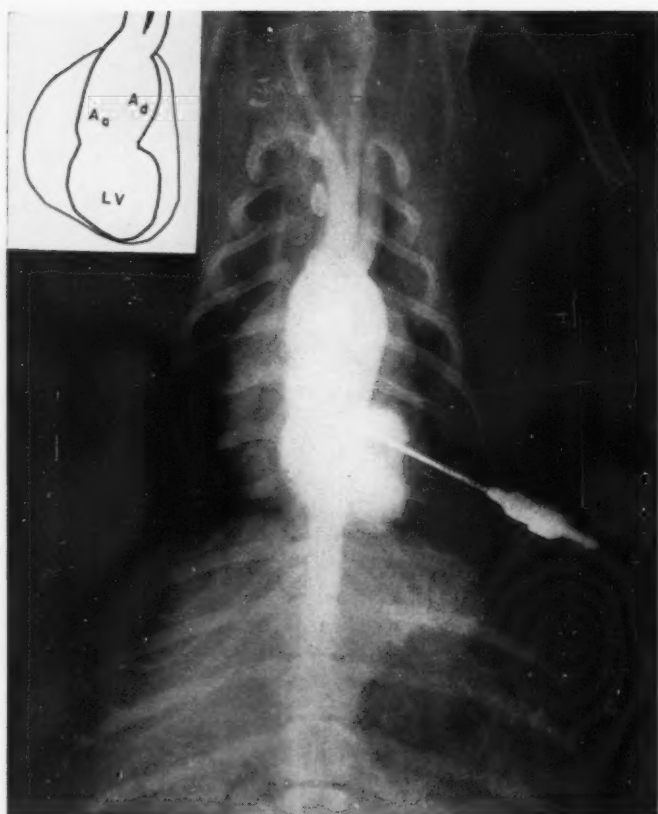


Fig. 6—Dorsoventral radiograph and schematic diagram of normal canine thorax with left ventricle (LV), ascending aorta (Aa), and descending aorta (Ad) opacified. Brachiocephalic and left brachial arteries are unlabelled cephalic extensions from the aortic arch.

the left atrium, posterior vena cava and pulmonary veins.

The aortic density may usually be visualized, leaving the dorsocephalic border, coursing briefly cephalad and dorsad, then reversing direction and proceeding directly caudad immediately ventral to the vertebra. The anterior vena cava and the posterior vena cava, respectively, may be seen leaving the dorsal cephalic and caudal borders.

Relationships of Chambers and Great Vessels to Each Other.—The crescent-shaped right ventricular lumen envelops the circular left ventricular lumen on the left cephalic, cephalic, and right borders (fig. 3a, 5a). Injection of radiopaque material usually opacifies the right ventricular lumen as if it is located only cephalad to the left ventricle and interventricular septum. The extension to the right of the

left ventricle is a relatively narrow portion of the crescent; therefore, it contains little radiopaque material (fig. 3b) and may appear angiographically "silent" (fig. 3a). The main pulmonary artery density leaves the right ventricle dorsad, coursing from the left cephalic extremity. It sends the left pulmonary artery caudad and the right pulmonary artery rightward (fig. 5b).

Out of the right cephalic portion of the circular left ventricular lumen, the ascending aorta courses cephalad and dorsad (fig. 4a, 6). The sinuses of Valsalva may be visualized as a dilatation at the base of the aorta within the center of the cardiac silhouette. Often, after left ventricular injection of the radiopaque medium, the left anterior descending coronary artery may be seen in the lateral radiograph leaving the caudal Valsalva sinus and coursing

ventrad along the left longitudinal groove which separates the right and left ventricles on the left septal border. The left circumflex coronary artery also may be seen leaving the caudal Valsalva sinus and extending over the caudal and dorsal border of the cardiac base. The right coronary artery is less often seen, but occasionally appears as a faint density leaving the cephalic Valsalva sinuses and coursing ventrad and slightly caudad.

Discussion

Radiographic relationships may be best described by the actual angiocardiograms; thus, the reader is referred to (fig. 4-6). These findings are in essential agreement with other investigators.¹ Correlation between these radiographic relationships and those obtained by anatomic cross-sections taken through the thoraces of 40 solidly frozen dogs will be presented in a later communication.

Summary

The right ventricle lies predominantly cephalad and slightly ventrad and dextrad to the left ventricle. The aortic shadow occupies the midline, bulging cephalad; the main pulmonary artery, forming the left cephalic cardiac border, lies sinistrad to the aorta.

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Five People Believed to Have Acquired Psittacosis from Birds

Five unrelated cases of psittacosis were reported in California. Two were in women who had parakeets in their homes. One woman had fever, headache resembling viral influenza, slight cough, pneumonitis, and a fourfold rise in antibody titer demonstrated by complement-fixation (CF) tests. The other woman had fever, weakness, dry cough, substernal chest pain on respiration, and CF test showing rise of titer from 1:32 to 1:64. The parakeets tested negative.

Two cases were in men who worked with birds. One was an animal caretaker at an experimental laboratory who regularly cleaned bird cages and fed doves. Doves were found to be positive for psittacosis. The other man had pet pigeons which were found to be negative.

The fifth case involved a 21-year-old girl who worked with psittacosis virus in a laboratory.—*Morbid. and Mortal. Rep., U.S. Dept. Health, Education, and Welfare*, 8, (Nov. 28, 1959): 2.

Adrenocortical Insufficiency

in a Dog

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George D. WEBSTER, JR., M.D.
Joseph F. SKELLEY, V.M.D.

WELL-DOCUMENTED cases of spontaneous adrenocortical insufficiency (Addison's disease) in dogs are absent from the veterinary literature. In 1953, a study of 3 dogs with adrenal cortical atrophy was reported in which clinical and laboratory studies were largely noncontributory.⁹ In 1958, 2 cases with chronic primary adrenocortical insufficiency were described.⁸ In 1 dog, antemortem diagnosis was based on clinical signs and eosinophilia unaffected by adrenocorticotrophic hormone (ACTH) as judged by Thorn's eosinophil-response test. The following is a report of the first clinical case of this syndrome reported in the United States.

Case Report

On Nov. 5, 1958, an English-born 4-year-old Welsh Corgi bitch (fig. 1) was referred to the veterinary hospital of the University of Pennsylvania.

Her illness began in April, 1958, with emesis and hemorrhagic diarrhea. Fever was absent and she appeared to recover fol-

lowing symptomatic treatment by a local veterinarian.

One month later, a second nonfebrile episode was characterized by anorexia, mild nonhemorrhagic diarrhea, asthenia, and polydipsia. The normal long hair had been replaced by thick fuzz. Soon, the dog became critically ill. Laboratory findings at this time were: blood urea nitrogen (BUN), 85 mg./100 cc.; serum sodium (Na), 120 mEq/liter; serum potassium (K), 8.5 mEq/liter. The owner, a physician, made a tentative diagnosis of adrenocortical insufficiency and gave 25 mg. of oral cortisone per day. Clinical signs abated and the dog seemed to recover. Maintained on this dosage, the dog remained well and ate heartily. Attempts at lowering the dose resulted in asthenia and anorexia. Normal estrus occurred one month prior to admission.

Past medical history indicated that the dog had been vaccinated against distemper with killed-virus vaccine in England and modified live-virus vaccine in the United States, and against hepatitis and rabies. She had a normal six-month estrous cycle with no history of pseudocyesis. Duration of estrus was three weeks. A litter was whelped without difficulty in the spring of 1957. There were 2 normal pups, 2 stillborn, and 1 pup was killed because of a hindleg deformity.

On physical examination, the dog appeared overweight, of quiet disposition and had a thick fuzzy coat. Except for tachycardia and slightly enlarged tonsils, she appeared in good health. Her weight was 24 lb.; temperature 102.0 F.; pulse rate, 140; and respiration rate, normal.

Cortisone administration was halted just

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The authors thank Drs. Joseph C. Touchstone, Department of Obstetrics and Gynecology, Hospital of the University of Pennsylvania, for the determinations of the urinary alpha-ketolic steroids; John G. Reinhold, William Pepper Laboratory, Hospital of the University of Pennsylvania, for the determination of protein-bound iodine; William Medway, clinical laboratory, School of Veterinary Medicine, University of Pennsylvania, for hematologic and biochemical determinations; and Donald Patterson, heart station, School of Veterinary Medicine, for electrocardiography and blood pressure studies.

prior to admission. Aside from an unusually quiet disposition and variable appetite, her hospital course during the first admission (Nov. 5 to 15) was uneventful. Initial and subsequent laboratory findings, on November 7, are summarized (table 1 and fig. 2). The only grossly abnormal finding was eosinophilia (16%). The results of two eosinophil-response tests¹⁴ are reported (table 2). Since a diagnosis of adrenocortical insufficiency seemed questionable at this time, it was recommended that cortisone therapy be temporarily withheld.

The owner reported that the dog had walked with difficulty on the day of discharge. On that same night, there was frequent vomiting (predominantly retching), complete anorexia, marked asthenia, and polydipsia. Serum sodium concentration was 120 mEq/liter. The owner administered several 25-mg. doses of cortisone *per os* over the next 48 hours. However, owing to persistent emesis, the quantity retained could not be estimated. Asthenia did not improve. The dog was admitted to the hospital a second time on November 17.

On physical examination, the dog was found to be severely depressed and ataxic. Eyes and membranes appeared normal. Abdominal palpation was negative. Temperature was 102.9 F., pulse rate, rapid, and respiration rate, normal. Mean systolic blood pressure (BP) was 140 mm. Hg.** Cortisone was purposely withheld to await

further developments, and the dog was placed in a metabolism cage. Significant laboratory findings (table 1) were: eosinophils, 21/100 w.b.c.; BUN 61.7 mg./100 cc.; serum Na, 120 mEq/liter; and serum K, 6.4 mEq/liter.

On the morning of November 19, the dog appeared to be in Addisonian crisis, showing severe prostration, dehydration, and anuria of 24 hours' duration. She refused water and her bladder was empty on palpation. Temperature was 99.1 F., pulse rate 48, and respiration rate, normal. The mean systolic BP was 80 mm. Hg. An electrocardiogram (EKG) (fig. 3) showed complete atrioventricular block, the atria and ventricles being activated by ectopic pacemakers. T-wave changes were suggestive of hyperkalemia. Bowel movements were absent, but the anal area was stained with watery feces. Significant laboratory findings (table 1) were: eosinophils, 28/100 w.b.c.; BUN, 86.6 mg./100 cc.; serum Na, 119.0 mEq/liter; serum K, 6.9 mEq/liter; and serum chlorides (Cl), 80 mEq/liter.

In spite of her collapsed state, the dog displayed a remarkably alert interest in a housefly which circled her head while she lay on a table. Several quick jaw-snapping motions were made in an obvious effort to catch the fly.

Prior to receipt of laboratory data, treatment with prednisolone (25 mg., i.m.), procaine penicillin (300,000 units), and 250 cc. of isotonic saline solution in 5 per cent dextrose was administered by intravenous drip. Following treatment, on the after-

**Blood pressure was measured by an Infraton Automatic Blood Pressure Recorder, Medical Electronics Development Co., Great Neck, N. Y.

TABLE 1 — Hematologic and Biochemical Values in the Blood and Serum of a Dog with Primary Adrenocortical* Insufficiency

Date	w.b.c.	Seg.	Lymphocytes	Monocytes	Eosinophils	Basophil	Hb. Gm.	BUN ¹⁰ mg./100cc	Na mEq/liter	K mEq/liter	Cl ¹⁸ mEq/liter	Sugar ⁷ mg./100cc.
11/7/58	10,300	71	11	2	16	..	16.0	26.2	148.0	4.2
11/10	13,800	67	23	7	3	..	15.0	28.0	136.2	4.7	93
11/18	17,100	60	12	5	21	3	61.7	120.0	6.4	128
11/19	13,400	44	25	3	28	..	14.4	86.6	119.4	6.9	79.9
11/20	80.4	103.8	8.1	76.2
11/21	28.0	125.0	4.7	90.4
11/22	138.7	4.3	96.4
11/24	141.2	4.2	108.3
11/25	138.7	4.3	103.7
11/26	70,700	86	10	4	6.7	13.1	143.7	5.9	57
12/1	15,100	85	9	6	8.4	149.5	4.4	112.0
12/4	13,900	97	3	7.6	163.8	5.6	121.2
12/8	147.5	5.5	109.2
12/10	10,400	79	12	8	1	..	12.4	141.6	5.4	106.5
1/16/59	18.7	149.9	5.4	110.2

*Chemical analysis for sodium and potassium by flame photometry.



Fig. 1—Normal appearance of Welsh Corgi with primary adrenocortical insufficiency when maintained on adequate steroid therapy.

noon of November 19, the animal seemed alert and much improved. Her temperature was 98.8 F. and pulse rate, 132. The EKG showed disappearance of heart block and hyperkalemia (fig. 3). Urine was voided and the bladder was filled. Further treatment consisted of 225 cc. of isotonic saline

solution in 5 per cent dextrose intravenously (i.v.). The dog continued to improve and by evening she ate salted horsemeat readily. Urine flow was now copious. Intramuscular hydrocortisone acetate, 25 mg., (later found to be several years outdated†) was given. The dog consumed 650 cc. of water during the afternoon and night.

On the morning of November 20, the dog was again prostrate, she was vomiting colorless mucoid material, and had bloody diarrhea. Her temperature was 98.6 F. and pulse rate, 56 per minute. Laboratory findings (table 1) were: BUN, 80.4 mg./100 cc., serum Na, 104 mEq/liter; serum K, 8.1 mEq/liter; and serum Cl, 76 mEq/liter, Hydrocortisone hemisuccinate, 20 mg. in

†In an attempt to test the potency of the expired hydrocortisone, a normal dog was injected with 25 mg. (i.m.). There was no significant change in the total or differential white blood counts three hours after injection. Similarly, there was no change in blood sugar at one, two, and three hours after injection. Eosinophil counts were not possible because the blood was accidentally collected in oxalate instead of heparin. There was insufficient hydrocortisone for a repeat test.

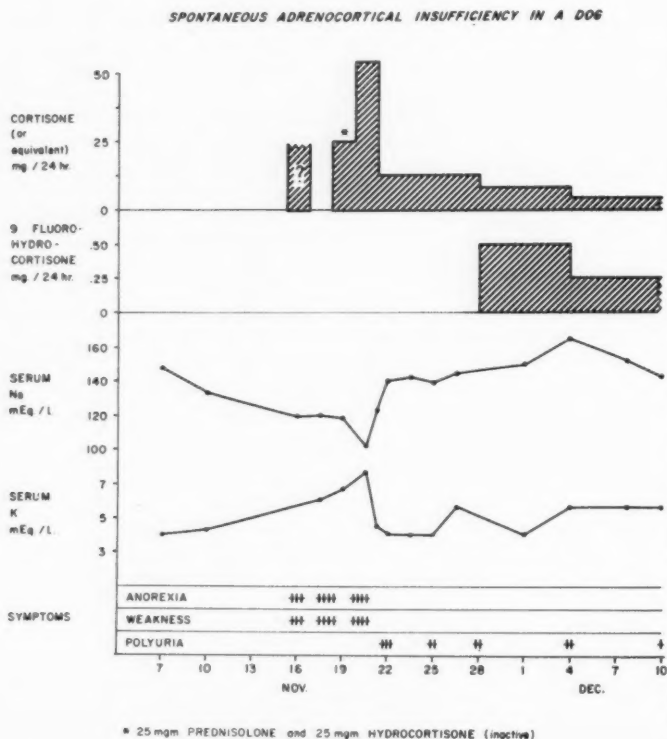


Fig. 2—Graph summary of laboratory findings of dog in figure 1, for November and December 1958.

TABLE 2—Results of Two ACTH Tests on a Dog with Primary Adrenocortical Insufficiency

	Nov. 8, 1958 (Eos./cmm.)	Nov. 11, 1958 (Eos./cmm.)
Before ACTH	1,631	6,937
1 hr. post-ACTH (30 u.i.m.)*	1,309	14,996
3 hr. post-ACTH (30 u.i.m.)	1,176	14,585
5 hr. post-ACTH (30 u.i.m.)	1,099	7,803
7 hr. post-ACTH (30 u.i.m.)	17,882

*Units intramuscularly

350 cc. of saline solution, was given i.v. and 25 mg. of unexpired hydrocortisone acetate and 300,000 units of procaine penicillin i.m. During infusion, the heart rate increased to 128/minute. The dog appeared more responsive and alert and voided pale clear urine. Although slightly ataxic, she could walk. She drank 950 cc. of water in four hours. By afternoon, the pulse rate was 150 and the mean systolic BP, 140 mm. Hg. Urine flow was copious. Another 10 mg. of hydrocortisone acetate was given intramuscularly, and 150 cc. of hypertonic saline solution (1.8%) was given intravenously.

On November 21, the animal appeared normally bright and walked steadily. Her temperature was 101.6 F., pulse rate 160, and respiration rate was normal. The BUN was 28.0 mg./100 cc.; serum Na, 125 mEq/liter; serum K, 4.7 mEq/liter; and serum Cl, 90 mEq/liter. During the night, 800 cc. of water had been consumed and

TABLE 3—Urinary Alpha-Ketolic Steroids*

Av. normal dog present study (day)	Total ketols 2.2 mg./24 hr.
	mg./24hr.
1	1.1
2	1.4
3 10 units ACTH administered	1.2
4 10 units ACTH administered	1.5
5 10 units ACTH administered	0.8

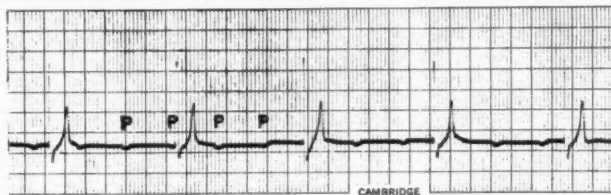
*Determination of urinary alpha-ketolic steroids was made by the blue tetrazolium reduction method of Touchstone and Hsu.²³ The urines were extracted with chloroform and ethyl acetate after incubation with β -glucuronidase for 48 hr. at pH 4.5 and a temperature of 37C. The organic phases were washed with alkali and water and then evaporated to dryness in vacuo. Using this method of quantitation, a normal response to corticotrophin administration will result in a two-to-threefold increase in the output of alpha-ketolic steroids in the urine as measured by reduction of blue tetrazolium. Table 3 indicates that the present dog showed urinary corticoid levels much lower than the average for normal dogs. The dog showed no response to the administration of corticotrophin.

another 400 cc. during the day. By 4:00 p.m., 1,500 cc. of urine was collected.

The dog seemed fully recovered and ate heartily. Intramuscular hydrocortisone acetate was continued at a level of 5 mg. three times a day (t.i.d.) On this regimen, she maintained apparent good health and fairly constant body weight (approximately 24 lb.). However, water intake and output remained high. Serum electrolytes returned to normal. Radiographic evidence of adrenal

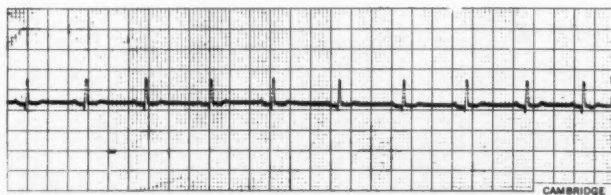
Fig. 3—A—Lead CV_{EL}. (Wilson central terminal paired with exploring electrode at the 6th left intercostal space at edge of sternum) of an electrocardiogram taken on Nov. 19, 1958, before treatment with saline solution and corticosteroids. There is complete atrioventricular block with ectopic atrial pacemaker (P waves are normally upright in this lead). Note that there is no constant relationship between the P waves and the QRS complexes. The atrial rate is 120/min., while the ventricles are beating at 45/minute. The high amplitude, peaked T waves are suggestive of hyperkalemia. Standardization: 1 millivolt = 1 cm.

A



B—Lead CV_{EL}. of an electrocardiogram taken on Nov. 21, 1958 (2 days after beginning treatment). There is normal sinus rhythm with upright P waves and T waves of normal configuration. Standardization: 1 mv. = 1/2 cm.

B



disease could not be demonstrated by radiograph of the abdomen on November 25. Other laboratory data obtained during hospitalization included: total serum proteins,²⁴ 9.0 and 7.3 mg./100 cc.;[‡] serum cholesterol,² 260 mg./100 cc.; serum magnesium,¹⁵ 1.25-2.15 mg./100 cc.; icteric index, 2 to 10 units; serum phosphorus,⁵ 2.3-4.9 mg./100 cc. Sedimentation rate was not elevated. Serum protein-bound iodine (PBI)¹ was 2.7 and 5.2 μ g./100 ml.[‡] Intradermal tuberculin and histoplasmin tests were negative.

A sudden marked neutrophilia was reported on November 26 (table 1). The total leukocyte count was 68,000 with 86 per cent neutrophils (79% segmented, 7% nonsegmented). In spite of normal temperature (101.7 F.), she was given chloramphenicol 100 mg. t.i.d. Her low hemoglobin (6.7 Gm./100 cc.) was attributed to frequent blood sampling for laboratory studies. In spite of marked leukocytosis and anemia, the dog appeared well. The leukocyte count gradually returned to normal (table 1). She was given several doses of crude liver extract with iron.

On November 28, injectable hydrocortisone was discontinued and the dog was switched to oral 9- α -fluorohydrocortisone 0.25 mg. and cortisone 6.25 mg. b.i.d. On this regimen, the serum Na rose to 164 mEq/liter; polydipsia and polyuria continued. Dosage of 9- α -fluorohydrocortisone was reduced to 0.125 mg. b.i.d. and cortisone to 3.12 (b.i.d.).

On December 6, serum Na was 147 mEq/liter; serum K, 5.5 mEq/liter; and serum Cl, 109.2 mEq/liter. Water intake was now 200 cc. and urine output 300 cc. in 24 hours. Leukocytosis and eosinophilia had disappeared.

On January 14, 1959, the dog was readmitted to the veterinary hospital for special studies. There were no abnormal findings on physical examination and her weight was recorded at 27 lb. Serum Na was 150 mEq/L; serum K, 5.4 mEq/L; serum Cl, 110.2 mEq/liter; and BUN, 18.7 mg./100 cc. Hemoglobin was 16.8 Gm./100 cc.

The dog was placed in a metabolism cage and her daily steroid intake was limited to 0.25 mg. of 9- α -fluorohydrocortisone. Daily 24-hour urine samples were collected in flasks containing 15 ml. of chloroform. After a two-day control period, 10 units of

ACTH were administered i.m. on three consecutive days. Three 24-hour urine samples were collected during this time. The daily urinary excretion of α -ketolic steroids was 1.2 mg./24 hours as compared with an average normal of 2.2 mg./24 hours.* There was no rise in urinary α -ketolic steroids following ACTH (table 3).

On January 20, the day of final discharge, mean systolic blood pressure was 170 mm. Hg.

On a home-maintenance regime of 0.125 mg. 9- α -fluorohydrocortisone and 6.25 mg. cortisone daily, the dog has remained well to the present time. Normal estrus occurred in April, 1959.

Discussion

Diagnosis of adrenocortical insufficiency was based on clinical findings: electrolyte abnormalities, eosinophilia, and low urinary excretion of adrenocortical steroids (measured as α -ketolic steroids); and response to steroid replacement therapy.

Clinical signs which develop in dogs following adrenalectomy are identical to those present in this case and closely resemble adrenocortical insufficiency (Addison's disease) in man.^{3,12,21}

While the adrenal cortex is known to elaborate many biologically active substances, all of the physiologic disturbances that develop in adrenal insufficiency can be corrected by the provision of compounds having two types of activity, i.e., glucocorticoid activity and mineralocorticoid activity. Compounds such as cortisone and hydrocortisone exert mainly glucocorticoid effects, but each has moderate mineralocorticoid activity as well. Aldosterone, on the other hand, has predominantly a mineralocorticoid action.

The features which result mainly from a deficiency of glucocorticoid activity are anorexia, impaired carbohydrate metabolism, and eosinophilia. Objective evidence of impaired carbohydrate metabolism was not observed in the present case but is usually manifested as a tendency toward fasting hypoglycemia. Abnormal utilization of glucose may account for some of the observed muscular weakness. Normally in dogs there is a 75 to 90 per cent fall in

[‡]Two values are determined for these instances.

*This average was based on 24-hour urine collections from 12 normal dogs.

circulating eosinophils after ACTH administration.¹⁴ In this dog, no significant change was evident at two, three, and four hours after the first test dose. The sharp rise in eosinophils after a second ACTH injection four days later is unexplained.

Deficiency of mineralocorticoid effects accounts for the marked asthenia, the cardiovascular effects, and for the azotemia, hyponatremia, and hyperkalemia. Aldosterone facilitates renal tubular reabsorption of sodium and secretion of potassium so that in its absence there is a tendency to sodium wasting and dehydration. Probably of more significance, at least in the development of the acute crisis, is the effect of the salt hormones in the internal distribution of electrolytes and water.^{6,11}

In acute adrenal insufficiency, there are marked transfers of sodium and water out of the extracellular compartment. The combination of these effects accounts for the marked hyponatremia and hyperkalemia and extracellular dehydration. As a consequence of the latter, hypotension develops, and the resulting fall in glomerular filtration rate contributes to the azotemia and may lead to anuria. In addition, the presence of the adrenocortical hormones appears to be necessary for the normal response of the vascular system to various pressor agents.

The cardiac conduction defects, with tall peaked T-waves, represents the effect of severe potassium intoxication. The blood observed in the feces at the height of the symptoms may have been the result of gastrointestinal ulcers. Such lesions are common in adrenalectomized dogs.¹⁶

One of the classical manifestations of Addison's disease in man is pigmentation from deposition of melanin in the skin and mucous membranes. Such pigmentation was not evident in this dog and usually does not develop in adrenalectomized animals.¹⁶

Adrenocortical insufficiency can be primary or may develop secondarily as a manifestation of panhypopituitarism. In the latter cases, there is evidence of deficient thyroid and gonad function. Serum PBI levels in the present case are believed to lie within the normal range for dogs, indicating normal thyroid function.¹³ Hypogonadism is ruled out by the presence of normal estrous rhythm before and after the onset of the disease. Moreover, the adrenals were unable to respond to exogenous ACTH administration. In man, a normal re-

sponse to corticotrophin is a two to three-fold increase in the urinary excretion of *alpha*-ketolic steroids.^{20,23} Finally, adrenal insufficiency secondary to hypopituitarism is usually not marked by severe derangements in salt metabolism, since aldosterone is believed to be largely independent of pituitary control.⁴ This case, then, can be classified as primary adrenal insufficiency.

The underlying cause of the adrenal insufficiency is unknown. In man, tuberculosis, tumors, amyloidosis pyogenic infections, histoplasmosis, coccidioidomycosis, and blastomycosis are all capable of destroying the adrenal cortex.^{3,16} The term idiopathic atrophy is applied to describe cortical destruction of unknown etiology. The absence of radiographic evidence of adrenal calcification and negative tuberculin and histoplasmin tests argue against, but do not rule out, tuberculosis, histoplasmosis, and adrenal tumors. Based on one report⁹ of 3 cases of adrenal atrophy in dogs, this would appear to be a strong possibility. A more definitive diagnosis will not be possible until necropsy at some future date.

Clinical signs which develop in dogs following adrenalectomy closely resemble adrenocortical insufficiency (Addison's disease) in man.

The minimal dose of cortisone compatible with normal health in this 24-lb. dog was 25 mg. per day. This is approximately 2.2 mg./kg./day and is in close agreement with maintenance recommendations for adrenalectomized animals.¹⁰ Cortisone 1.86 mg./kg./day, is the minimum level at which adrenalectomized dogs on a low sodium diet can maintain health, vigor, and a nearly normal electrolyte pattern.¹⁹ While cortisone alone can be used for maintenance therapy, in this case the stimulation of appetite by high dosage levels made control of obesity as difficult as in human patients.¹² Excessive mineralocorticoid effects in dogs result in hypertension, hypernatremia, and a diabetes insipidus-like state.¹⁷ These complications were apparent during

the period of hospitalization (table 1 and fig. 2).

The final choice of corticoids for long-term maintenance in this case was based on the premise that a combination of 9- α -fluorohydrocortisone (principally a salt hormone) and cortisone (principally a sugar hormone) would provide good regulation at relatively low total dose levels. In severe stress situations, the cortisone should probably be increased to avoid exacerbation of acute hypocorticalism.

Summary

In a case of primary adrenocortical insufficiency in a 4-year-old dog, most classical manifestations of Addison's disease were present. These included anorexia, emesis, asthenia, cardiovascular effects, eosinophilia, dehydration, anuria, azotemia, hyponatremia, and hyperkalemia. These effects can be explained on the basis of a deficiency of the glucocorticoid and mineralocorticoid hormones of the adrenal cortex.

Response to replacement therapy (cortisone and 9- α -fluorohydrocortisone) was dramatic, resulting in complete reversal of all abnormalities. The maintenance dose of oral steroids is in close agreement with the dose required to maintain adrenalectomized dogs.

It is concluded that the adrenocortical insufficiency is primary, rather than secondary, to panhypopituitarism. Administration of ACTH failed to elicit an eosinopenic response and did not increase the low level of urinary α -ketolic steroids; thyroid function and gonad function were normal.

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Editorial

Solving the Problem of *Leptospira pomona* Diagnosis

Because of clinical diagnostic difficulties encountered in determining presence of *Leptospira pomona* infection in cattle, serological tests have been developed and utilized.

However, a single serological test has little value where an individual animal is concerned. *Leptospira pomona* serum antibodies may persist for years, so that it is difficult to tell from test results whether infection occurred recently or at some time in the distant past.

Serologic testing of an individual is of greatest value when a second test is conducted 2 to 4 weeks after the first. If, on the second test, the titer is markedly higher, infection is recent. Diagnosis of recent infection indicates that associated susceptible animals should be vaccinated. Since susceptible animals are difficult to single out, vaccination of the entire herd may be the only practical solution.

The main disadvantages of this diagnostic procedure result from the delay in confirming the diagnosis imposed by waiting 2 to 4 weeks to find out if the antibody titer changed. By the time a diagnosis is confirmed, many more animals may become exposed, reducing the value of vaccination and possibly subjecting the herd owner to further losses. In addition, there is the cost of making two calls to draw blood samples and possibly a third to vaccinate the herd.

Herd diagnosis, rather than individual diagnosis, provides a better approach to

leptospirosis control because serologic test results from all the animals in a herd or from a representative sample (20 to 50% of the herd) give a far better idea of the status of *Leptospira* infection than serologic test results from individuals.

Since tests of recently infected herds usually show a preponderance of high titers (1:160 or greater on the plate (P) or capillary tube (CT) test and 1:10,000 or greater in the agglutination-lysis (AL) test), a single test often provides sufficient information to enable the practitioner to recommend a course of action. If only 1 or a few animals have high titers while others have low titers (1:40 P, CT), the herd probably has experienced recent infection and vaccination of susceptible animals should be recommended. In herds where titers do not exceed 1:40, recent *L. pomona* infection is not generally considered, even if there is leptospirosis in the area and clinical signs are observed.

The matter of significant titer is a controversial one. However, the serologic titer of 1:10, as used in the screening plate test, should never be interpreted to indicate presence of leptospiral infections. Titers of 1:10 and even 1:40 may be nonspecific and possibly indicate false positives. Some laboratories have reported results as positive on the basis of the 1:10 screening test without stating this fact. For practical purposes, 1:160 (P, CT) or 1:10,000 (AL) seems a justifiable infection titer. Even at a titer this high, there is the possibility that the serologic reaction is due to leptospiral strains other than *L. pomona*. This problem of cross-reactions remains unsolved.

Diagnosis on the basis of clinical signs alone recently has been shown to have a good deal of validity. In the JOURNAL of the AVMA for June 1, 1959, R. K. Jones, at Purdue, showed that Indiana practitioners could diagnose leptospirosis on the basis of clinical signs in a significant number of cases. Judging by this report, one could conclude that, in areas where leptospirosis was known to occur, a vaccination program instituted immediately following a clinical diagnosis of leptospirosis might be advisable. However, the decision to vaccinate on this basis depends on the clinician's judgment of the advantages of quicker protection resulting from imme-

diate vaccination and on his judgment of the cost of vaccination compared with the cost, inconvenience, and delay associated with blood sampling and serologic diagnosis.

Serologic diagnosis has no substitute as

a basis for individual, herd, or area leptospirosis control. Yet, practicality may dictate that it be by-passed in some instances. In areas where leptospirosis has not been known to occur, serologic diagnoses should always be utilized.

Hog Cholera Eradication Possible

Ninety-six per cent of all garbage fed to hogs is cooked. More will possibly be cooked this year.

Hog cholera can be eradicated by reporting all cases to state officials, by treating

infected herds, by vaccinating all herds in the vicinity, and by tracing the source and spread of each occurrence of the disease.—

F. J. Mulhern in Auburn Vet. 16, (1959): 19.

Map showing states which prohibit use of virulent virus for hog cholera vaccination



U.S.D.A. Agricultural Research Service

By Nov. 30, 1959, 32 states had laws prohibiting the use of virulent hog cholera virus.

from the *Research Journal*

Diabetes, Obesity, and Pyometra in The Dog

In a statistical review of necropsies on 10,993 dogs, 971 were obese, 487 had pyometra, and 167 had diabetes mellitus. Obesity, with an incidence of 11.6 per cent, was the most frequent disease of dogs in this series.

Obesity tended to occur at a slightly earlier age than diabetes mellitus and pyometra. Significantly greater numbers of females than males were affected by obesity and diabetes mellitus. Analysis of breed distribution in relation to the general dog population revealed that certain breeds were

significantly predisposed or resistant to diabetes mellitus, obesity, and pyometra. Predisposition or resistance to any of the 3 diseases was usually associated with predisposition or resistance to all 3 of the diseases.

The etiologic implications of this diabetes mellitus-obesity-pyometra syndrome are discussed.—Lennart Krook, Stig Larsson, and James R. Rooney: *The Interrelationship of Diabetes Mellitus, Obesity, and Pyometra in the Dog*. *Am. J. Vet. Res.*, 21, (Jan., 1960): 120-124.

NDV Defense Mechanism of the Chicken

Newcastle disease virus, when introduced into susceptible fowl, rapidly spread from the site of inoculation and could be detected in almost all tissues from 14 sites in the body within 48 hours and in all the tissues within 72 hours.

Virus titers for 8 tissues were obtained at 6-hour intervals. The increase in virus titer of the invaded tissues was usually interrupted at about the 36th hour, and this interruption lasted 12 to 24 hours. After the period of interruption, invasion of the central nervous system occurred and signs of disease became evident.

Injection of hydrocortisone usually elim-

inated the period of interruption and shortened the period between injection and appearance of clinical signs and eventual death.

The temporary interruption of the increase of virus titer in the tissues of infected chickens may be the result of a partially successful adaptation response of the host to the introduction of a foreign protein.—[Mary K. Asdell and R. P. Hanson: *Sequential Changes in the Titer of Newcastle Disease Virus in Tissues—A Measure of the Defense Mechanism of the Chicken*. *Am. J. Vet. Res.*, 21, (Jan. 1960): 128-132.]

Changes in Tongues of Cattle with Vesicular Stomatitis

The morphogenesis of lingual lesions of vesicular stomatitis in cattle was determined by correlation of their gross, subgross, and microscopic characteristics. Lesions caused by Indiana and New Jersey types of virus were studied 48, 72 and 96 hours postinoculation.

The initial effect of vesicular stomatitis virus upon the bovine lingual mucosa was the development of blanched, slightly ele-

vated lesions characterized microscopically by intercellular edema, necrosis of epithelial cells, and inflammatory cellular infiltration.

Less than 30 per cent of the total initial lesions on 20 tongues inoculated with 2 strains of the virus developed into grossly evident vesicles by separation of the diseased mucosa from the underlying tissue. All vesiculation occurred within 48 hours postinocu-

lation. Vesicles were prone to occur at locations where there was friction between the tongue and the teeth or the dental pad.

The large percentage of initial lesions that failed to undergo gross vesiculation dehydrated *in situ* by seepage of the intercellular edema through the stratum corneum, and eroded in the manner of a dry, necrotic, mucosal lesion. The total pathologic picture of the bovine tongues observed 2, 3, and 4 days postinoculation is described and illustrated.

These studies developed a revised concept of the lingual pathology of vesicular stomatitis. The low percentage of vesiculation, as well as its transitory nature under controlled conditions of infection and observation, confirm clinical findings in recent field outbreaks of vesicular stomatitis in cattle.—[H. R. Seibold and James B. Sharp: *A Revised Concept of the Pathologic Changes of the Tongue in Cattle with Vesicular Stomatitis*. *Am. J. Vet. Res.*, 21 (Jan., 1960): 65-51.]

New Books

Handbook of Physiology

The second volume of the multiple-volume Handbook of Physiology is now available. As reported in a previous review (*JOURNAL*, Nov. 1, 1959: 494), the aim of the American Physiological Society is to provide a comprehensive survey of modern concepts and experimental findings pertain-

ing to physiology through a plan utilizing section editors and authors of international repute.—[*Handbook of Physiology*. Vol. 2. Edited by John Field, H. W. Magoun, and Victor E. Hall. 640 pages. Illustrated. Williams & Wilkins Co., Baltimore 2, Md., Price \$20.00.]

Lymphocytes and Mast Cells

This is a detailed discussion of lymphocytes, plasmacytes, mast cells, and certain other cells from the standpoint of their functions in supplying nutritive materials and regulating protein metabolism in the tissues. The inter-relationships of the nucleus, cytoplasm, the nucleolus and functions of the cell organelles in intracellular protein synthesis and storage is considered. Variations with respect to species, location, and physiologic or pathologic state are presented.

The chapter titles—Lymphocytes and Plasmacytes, Mast Cell Inter-relationships, Physiological Distribution of Mast Cells, Histamine, Effect of Mast Cell Products on Capillary Permeability, Heparin and Hyaluronic Acid, The Thymus, The Spleen,

Bone Marrow, Intestinal Lymphoid Tissue Relationships, and Inflammation and Wound Healing—convey the scope of the work. More than 1,100 literature references are listed and the citations in the context are clear.

A morphologist might wish for more illustrations but the authors clearly had in mind a dissertation of function rather than morphology. The book is fascinating because of its well-conceived organization and the lucid portrayal of a complicated subject. It will be useful to anyone concerned with interpretation of tissue reactions.—[*Lymphocytes and Mast Cells*. By M. A. Kelsall and E. D. Crabb. 399 pages. 31 illustrations. Williams and Wilkins Co., Baltimore, Md. 1959. Price \$8.00.]—CARL OLSON.



Picture Study of the Equine Practitioners' Program

First Day

To adequately report the American Association of Equine Practitioners meeting in Chicago, Dec. 14-16, 1959, the following quotations were adapted from AAEP's transcript. Accompanying snapshots show the various speakers in action.

A resume of the meeting, including the Association's newly elected roster, was published in the Feb. 1, 1960, issue of the JOURNAL, pp. 143-144.



The AAEP's registration desk was a busy place on December 14, the first day of the meeting.

Dr. Thomas E. Dunkin, Chicago, Ill., is shown facing the camera.

March 15, 1960



Another view of the AAEP's registration desk and this time Dr. Marion L. Scott is pictured facing the registrants.

The "P" for Practitioners (above) shows Drs. Wayne O. Kester, 1959 AAEP president, and Jordan Woodcock, AAEP's 1960 president (right).

Dr. Jordan Woodcock:

The pertinent question is: Where does therapy stop and stimulation begin? It seems that everybody here is in perfect agreement that stimulation, as such, is a bad thing and that the crux of the whole situation lies in the inferences and the inability of a practitioner, a trainer, or anyone else to function in an ethical manner and still abide by the 48-hour rule. Consequently, we would be much better off without a rule of such a nature than trying to function with it.

> > >

Mr. Marshall Cassidy



A rule that is gaining favor, much to my distaste, is one that prohibits the administration of a drug 48 hours before a race. I do not believe it is possible to determine whether a drug was given within 48 hours rather than 50 hours.

I do not believe we will ever have a satisfactory rule unless veterinarians and horsemen are able to give to horses such aid as they deem necessary to provide a healthy racing animal.

Basically, I am strongly opposed to any time limit on the administration of medication. I am in favor of leaving the task to the stewards to supervise it, to the extent that veterinarians could treat a horse properly without attempting to affect his racing speed or condition.

The Racing Commissioners are in favor, at the present time, of a 48-hour rule. However, we are open-minded, we want to learn, and we want to find out what the views of the AAEP are. There has been a lot of talk here about protecting the owner by medicating a horse to bring him up to his true worth. I would just like to ask: Two horses have equal potential. They go into a race. Everything is equal, including the riders and all the other factors, but one horse is sound through his natural condition and the other is sound due to a drug. Which horse actually deserves to win the race? Shouldn't we also consider the investment that some other owner has in the naturally sound horse?

Mr. James H. Inglis



Mr. Thaddeus B. Bruno



We in racing are very much concerned about permitting any one individual to have the right to do anything which might alter the racing form of an animal. I am not saying that it is wrong to give an ailing horse something that will help him. I do say that it is wrong to let a trainer, veterinarian, or layman have the right to determine whether it shall be given or not, to the extent that it affects the horse's performance (and there is a big difference).

The person wagering assumes that the animal is a healthy animal and that the animal is qualified to start, and that the animal will run consistently to his or her former form. If that form can be altered at the will or pleasure of any individual or any entity, then the information which the public has is not completely factual. Whether the 48-hour rule solves the problem, I cannot completely say. But it was with that thought in mind that we established a 48-hour rule.

Should any horse ever run under any drug of any kind or at any time? My own feeling, and I think that of most of the chemists in our group, is this: Any drug can or may affect the performance of a horse. We would not like to point at the drug and say it is a stimulant or depressant. Almost any drug can have effects other than the ones you may be familiar with.

We must agree that the use of pain-relieving drugs will change the performance or the activity of an animal. As soon as we relieve pain, we have changed his performance. That does not mean we have eliminated the underlying cause—the lesions there in the tissues. It means we have more or less masked it and, obviously, the underlying change may still be present, or may be present in a more aggrieved form after treatment.

I think the horse running with lesions present, or with abnormal tissues or tissue reaction present, certainly should not be on the race track.

The remarks I have made about pain relievers can be made about almost any drug that is used. This leads me to say that no drug should be given to any horse before racing. This is a basic pharmacological fact we can't avoid: that there is going to be some influence exerted by any drug used.

Dr. Y. T. Oester



Mr. John Manfuso:

We all know that there are cases of undetected stimulation occurring, even though the percentage is relatively small. The H.P.B.A. has gone on record and is unalterably opposed to any medication or stimulation which will artificially improve the running ability of a horse beyond his normal capacity.

We are certain that your Association, the AAEP, is of a similar mind. However, I have heard the argument that some veterinarians feel that if they don't do the job, the trainer will do it anyway, so why not pick up the fee? I can't go for it, gentlemen, any more than I could go for anyone who would sponsor a boxing match and have one contestant with brass knuckles. It is absolutely contrary to any principle of fair play and sportsmanship. To me, it makes one almost an accomplice of moral thievery. I am certain you agree with me.



Dr. S. F. Scheidy:

There were 341 original articles on horses published in the last 10 years in five principal veterinary journals in this country. Of these articles, 60 per cent appeared in the semimonthly JOURNAL of the AVMA and the bimonthly American Journal of Veterinary Research. All of you who are specializing in equine diseases are urged to publish your work. Students often say, "Where can we get new information in diseases and treatment of horses? The textbooks are too old."

Dr. William O. Reed:

Any horse in training should be considered an athlete and should be given every opportunity to perform according to his normal ability. One of the greatest tennis players we ever had in this country was a diabetic. He was given insulin constantly, shortly before he played. That is just a simple example.

It is my feeling that sooner or later these medication rules will have to be altered to fit our present situation. It may take time, and we may have to do a lot of work.



Dr. M. B. Teigland:

Our major problem with horses could be classified into two groups. One of the most important problems is the treatment of inflammation. This covers many types of inflammation, whether produced by infection, by trauma, or by various and sundry accidents that occur to the horse.

We also are faced with the problem of treating deficiencies in the horse that is not doing well. We make every effort to determine why this horse is not doing well, and we make every effort to correct the deficiency.

I see no reason, in my own personal opinion, why it makes any difference whether this deficiency need be corrected over the period of a month before he races, if it is possible to do so, or whether this deficiency is one of such nature that it must be corrected within a reasonable time before he runs.

Second Day

Dr. C. J. Anderson:

Our own clinical experiences with equine infectious anemia and our conversations with other equine practitioners lead us to wonder how often this disease is being overlooked.

It seems to me that the test developed by the workers at the University of Miami for this disease has merit, but more work should be done to determine its reliability. Someone needs to find a way to finance this work.





Dr. W. F. Riley:

For relief of colic pain not too severe, I still like chloral hydrate sometimes mixed with potassium bromide. Demerol is preferred in the more violent forms of colic. It is not only spasmolytic, but also sedative and analgesic. For evacuation of the digestive tract, mineral oil is the safest thing we can use in horses. I prefer to augment this with a cathartic, such as a cascara sagrada preparation, given 2 or 3 days later.



Dr. G. H. Keown:

My theory is that "tying up" is brought on by a spasm of the blood vessels of the muscles. The muscles contract. Then, when the horse is exercised, less blood gets to the muscles at a time when more is needed. This interferes with physiological processes at the end-plate. Additionally, there is an increase in lactic acid, which accumulates in the muscle cell.



Dr. J. D. Wheat:

If you feel that surgery isn't actually necessary in order to save the life of the horse with colic, don't do it. Of course, colic accompanied by such conditions as intussusception, strangulated hernia, some types of impaction, and strangulated small intestine, must be treated surgically.

All horses with colic should be watched closely and the surgery, when indicated, should be done promptly.



Dr. O. R. Adams:

The incidence of thromboembolic colic, caused by embolism of the mesenteric arteries, is increasing with the increase in horse population. In the mild forms, the colic persists for several days to 3 weeks, and the horse isn't violent.

There is often atony of the bowel and no passage of feces, but there is no distention. By contrast, there is marked gaseous distention in obstruction colic. For some reason, this type of colic often follows worm treatment by 7 to 21 days. We see most cases of impaction in Shetland ponies on alfalfa, which is a poor horse feed.



Dr. Jacques Jenny:

I would recommend as an approach to dealing with equine fractures occurring on the race track the following: (1) Insurance underwriters should make horses available for treatment rather than have them destroyed. The insurance companies are owners of all horses that we would like to work on. If the horses are destroyed, the material on which we could gain our experience is lost.

(2) An agency should be formed which will underwrite the cost of treatment of equine fractures at the track. Funds for such an agency may be secured privately, and any possible revenue from disposal of horses could be channeled to this agency.

(3) I propose that a committee should be appointed by the AAEP to discuss and correlate efforts made in different parts of the country to achieve these ends.

Dr. Oscar Sussman:

I suggest we might do well, as a group, to cease using the term, "eastern equine encephalomyelitis." It is somewhat of a misnomer. We might get away from the onus, as far as the horse is concerned, if we use the term, "eastern encephalomyelitis".

If there are mosquitos and wild birds in an area, you have the vector of encephalomyelitis and a susceptible population. Horses should be vaccinated at least once a year; some believe it should be every six months. This vaccination is for the protection of the horse only and not a public health measure to protect man.



Dr. Arthur H. Davidson:

If, when dealing with equine dystocias, straining cannot be readily controlled and anesthesia is not desirable, the use of a trachea tube will help. It reduces the horse's ability to strain and, consequently, will save wear and tear on the operator's arms.

If, in dystocias, repulsion and dissection of the foal is necessary, it is usually advisable to anesthetize the mare. As a result, her help in delivery will be lost; however, the constant straining which may cause damage will be avoided. A mare in labor can absorb unusually large amounts of anesthetic. Damage to mares cannot always be treated immediately. It may be that vulver tears may have to wait until the next day before they can be sutured. Cervical lacerations may look terrible the week following injury, but, if the muscles are not severed, they usually heal satisfactorily.



Dr. Charles Raker:

I feel that an arthritic joint should not be injected blindly with an anti-inflammatory agent especially if there is a possibility that one may want to perform surgery at a later date. The anti-inflammatory agent eliminates the pain and permits the horse to run, but if a bone chip is present, it continues to irritate and cause damage to the joint. Then there will be considerable proliferative change or a degenerative carpalitis which may become extensive. By this time, surgery will no longer be an effective approach.

In cases of fresh, uncomplicated chip fractures, one may expect 75 per cent recovery. Complicated chip fractures in which there is considerable degenerative change will result in about 50 per cent recoveries. Uncomplicated slab fractures can be expected to allow about 80 per cent recovery to racing soundness. Complicated slab fractures with involvement of the joint spaces will result in about 50 per cent recoveries. Where there is degenerative carpalitis, with changes around the margin of the joint, the prognosis is poor in 50 per cent or more of the cases.

This type of condition often recurs.

Left to right—Drs. William O. Reed (refer to page 287 for an abstract of Dr. Reed's remarks) and Charles Raker (see above).



Dr. Dale K. Sorenson:

In one limited epizootic of leptospirosis involving a herd of 54 Shetland ponies, a yearling filly became acutely affected. Clinical signs included marked depression, anorexia, icterus, fever, and bilateral iridocyclitis.

The animal was treated with penicillin and streptomycin parenterally for 7 days and was given supportive therapy consisting of intravenous fluids. Topically atropine was given to dilate the iris and chloromycetin solution to control secondary infection. The filly made a complete recovery.



Dr. D. L. Proctor:



I do not hesitate to make a large enough skin incision to give me a good view of the region on which I am working. I utilize multi-layer openings and multi-layer closures. I think one of the keynotes to good healing is a multi-layer closure that obliterates all air space.

Invariably, when one performs surgery on a joint, conditions are considerably worse than the radiographs indicated. This is a strict rule of thumb. The type of tourniquet I use is simply a piece of rubber innertube that I cut round and round. I believe it is important to keep blood out of a leg when surgery is being performed. I use a 4-inch bandage at pressure points, and I ligate the large vessels. This procedure permits a good clear field of operation, and I am able to see exactly what the extent of the pathologic conditions are.

Third Day

Dr. James T. O'Connor:

We are today medicating horses, often without proper diagnosis, due to the frame of mind of the trainer and the owner who might bring excessive demands which may not be for the benefit of the horse. The high pressures of business many times do not allow us to devote a proper amount of time to the individual.

Many of us are pressured into medicating horses by people whose knowledge of the drugs is frightening. Pecuniary reasons augment this trend. As practitioners and clinicians, we must not overlook the fact that we deal with objective clinical signs and with these alone in determining the course of treatment.



Dr. Bernard Brennan:

I have been fortunate that most horses that I have treated for purpura hemorrhagica have recovered. I have used blood transfusions, and I think they are beneficial. But the preferred treatment, in my opinion, is a very old one: formaldehyde in 5 per cent solution given intravenously.

I do not believe I would attempt to treat a horse for laminitis unless I used autohemotherapy. It simply involves removing 200 to 250 cc. of blood from the jugular vein and then injecting it in 10 to 15 cc. doses intramuscularly at various points in the animal's body.

Dr. R. R. Marshak:

A condition in man called paroxysmal paralytic myoglobinuria in every respect resembles the "tying up" syndrome of horses. The answers to the many questions about this disease are likely to come from detailed studies of the muscle masses, using biopsy and various chemical techniques. It is risky to think only in terms of therapy when we have little basis for understanding the problem.

Left to right—Drs. Bernard Brennan and R. R. Marshak.

Dr. John R. Steele:

Since the advent of all-weather race tracks which permit racing from March to December over a 2/5-mile course with 2-year-old pacers and trotters, it has become a great challenge to keep the Standardbred race horse sound, particularly in the rear quarters. The drive or push derived from the hind leg of the trotter or pacer is the site of the primary trouble in many horses. Quarter cracks are quite common in Standardbred horses running on hard tracks. Quarter cracks are best treated by using a hoof knife to cut them out up to the coronary band hair line and relieving pressure over the area using a bar shoe pad. Ichthammol packs and hot epsom suits applied to the affected area help reduce the soreness.



Dr. R. S. Lundvall:

I feel that better than 50 per cent of the colic cases that we see in Shetland ponies are due to verminous aneurisms. Of the fatal cases we see, nearly 100 per cent are due originally to verminous aneurisms. It seems that most cases of strangulations, intussusceptions, and volvulus are primarily due to aneurism in the middle mesenteric artery. Apparently, this causes enough impediment to blood supply so that an irritation of the gut occurs.

We use a stomach tube and, in some cases, a dose syringe for oral administration of medicaments to Shetland ponies. Passing the stomach tube in a Shetland pony is a little difficult and occasionally the tube may be passed in one nostril and come out the other. But a balling gun can be even more embarrassing because a 240 grain bolus may not even pass through the small esophagus of a Shetland.



Dr. D. K. Detweiler:

The diagnosis of heart disease in horses should not be based on murmurs. Unless the murmur is intense enough to cause a thrill, we have abandoned this as a reliable sign. Too many times I've detected a distinct murmur in the evening and have returned the next morning only to discover it had disappeared. The horse is the only animal in which this occurs.

Dr. E. A. Churchill:

When they start in training, many colts have excess fat on their bodies. Perhaps this results in some damage that isn't apparent at the time, yet has a bearing on whether "tying up" will occur later in life. I do feel that "tying up" is related to azoturia—that it's a matter of degree.



Mr. Clarkson Beard:

The equine practitioners can be the Grayson Foundation's best ambassadors. If we have a research program that meets the need, you are the people who are going to put it to use. We need your financial support, of course, but we want you to urge your clients to support our research program too.

Mr. Warren Wright:

As a representative of the underwriters of Lloyd's of London, I think the idea proposed for making fracture cases at the race track available for veterinary treatment may solve a problem that faces all of us.

The problem of the underwriters is that they have been losing money due to fractures and the resultant destruction at the race tracks. Our basic problem lies in the fact that the policy as written today is a death policy. It provides for destruction for humane reasons or natural or accidental death. There is no give or take in the policy.



New Animal Medical Center Now Being Built

NEW YORK CITY, N.Y.—Construction of the Animal Medical Center started on Jan. 4, 1960, on a site located on the East River Drive between 61st and 62nd Streets, in New York City, near Rockefeller Institute, Cornell Medical Center, and the Memorial Cancer Center. The Center was formerly known as the N.Y. Women's League for Animals.

It has received the endorsement of the Department of Health, Education, and Welfare and financial assistance for the development of its research program in the Center's new building which will contain both the Speyer Hospital for Animals, which will occupy the first floor, and the Caspary Institute for Veterinary Research.

Upon recommendation of the National Advisory Council on Health Research Facilities, the surgeon general has approved the Center's application for a public health service grant (not to exceed \$200,000) entitled the "Margaret M. Caspary Institute for Veterinary Research".

The new building will consist of seven floors above a parking area and lobby at street level; total area will be 70,000 sq. ft. The first floor (14,500 sq. ft.) will contain the new hospital which will accommodate the same number of patients as the present building now at 350 Lafayette St., in New York City, but will have amplified technical facilities and equipment for the intensive study of individual cases for research.

The second floor will be devoted to adoption and educational services, while the third floor will be used for administration. The third floor will also contain an auditorium, large enough for 100 persons, and a medical-research library.

The four upper floors will be devoted to formal laboratory research; 16,100 sq. ft. of this area to be developed at once and the remaining 9,000 sq. ft. to be reserved for future needs.

The remainder of the building above the hospital floor will be set back 40 feet in order to allow for additional construction when more space will be required for research projects.

AAHA to Meet This April in Boston

The American Animal Hospital Association announces that its 27th annual meeting

will be held at the Statler-Hilton Hotel, Boston, Mass., April 19-22, 1960.

The meeting will consist of one-half day of workshop seminars, and two and one-half days of scientific sessions designed for the practitioner of small animals. Demonstrations will be telecast by means of closed-circuit television through the courtesy of Pitman-Moore Company. A post-convention trip to Bermuda has been scheduled.

All veterinarians and their wives, for whom a social program is planned, are cordially invited to attend all sessions. Programs and hotel reservation cards will be available about March 15. For further information, address: Dr. Frank R. Booth, Executive Secretary, American Animal Hospital Association, 3920 E. Jackson Blvd., Elkhart, Ind.

Among the States and Provinces

Colorado

DENVER—NEW ROSTER.—Officers of the Denver Area V.M.A. are as follows: Drs. V. D. Stauffer, president; Richard Tolley, president-elect; J. D. McCluskie, vice-president; and Charles Garvin, secretary-treasurer.

S/CHARLES H. GARVIN, Secretary.

Florida

SARASOTA—DR. E. F. THOMAS TO SERVE EXAMINING BOARD.—Dr. E. Fred Thomas (GA '26) of Sarasota has been appointed a member of the Florida Veterinary Examining Board by Governor Collins, for a term of four years.

Dr. Thomas fills the vacancy created by the retirement of Dr. E. L. Matthews (MSU '39), after ten years of service.

S/M. W. EMMEL, Executive Secretary to the Florida V.M.A.

Kansas

MANHATTAN—K.S.U. HOLDS VETERINARY MEDICINE BUSINESS MEETING.—A veterinary medicine business conference was held on Kansas State University's campus, December 19.

Among the participants included on the program and their respective topics of dis-

cussion were: Mr. John H. Cooter, district manager of the social security administration, Topeka—the veterinarian and social security; Mr. Raymond Algott, manager, credit bureau, Manhattan—the use of credit bureau and billing; Mr. Donald Clingenpeel, audit division, internal revenue service, Wichita—federal income tax; Dr. A. H. Quin, Kansas City, Mo.—veterinary public relations; Mr. Ambrose Johnson, attorney, Manhattan—your will; Dr. Jim Davis, Kansas City, Mo.—the veterinarian in a commercial world; Mr. Keith Hayes, Charter Life Underwriters, Mutual of New York, Wichita—insurance and liability; and Dr. Warren J. Kilpatrick, Mediapolis, Iowa—evaluation of a practice.

Michigan

EAST LANSING—DR. STEELE HONORED BY THE USPHS.—Dr. James H. Steele, chief veterinarian of the United States Public Health Service's Communicable Disease Center in Atlanta, Ga., was honored by Michigan State University as a prominent alumnus of its College of Veterinary Medicine.

He was presented with a scroll at a dinner commemorating the 50th anniversary of the founding of the College, on January 20. Dr. Steele, an M.S.U. graduate—class of 1941, was one of six veterinarians honored on this occasion.

Ohio

COLUMBUS—AN EVENING ON COMPARATIVE CARDIOLOGY.—The Fifth District V.M.A. has planned an outstanding event for April 13, 1960. In cooperation with the College of Veterinary Medicine at Ohio State University, an entire evening will be devoted to "Comparative Cardiology."

This program will be inter-professional as each member is encouraged to invite a physician friend or any other member of the medical team to the meeting. It will be held in Sisson Hall at 8:00 p.m.—*The Ohio Veterinarian*, 8, (Jan., 1960): 25.

State Board Examinations

IOWA—May 31-June 1, 1960, Des Moines, Iowa. Applicants must be in the office of the Division of Animal

Industry, State House, Des Moines, not later than 8:00 a. m., on May 31. Additional information may be obtained by writing: Dr. A. L. Sundberg, Chief, Division of Animal Industry, State House, Des Moines 19, Iowa.

NORTH CAROLINA—June 20-22, 1960, Grove Park Inn, Asheville, N.C. Dr. James I. Cornwell, Secretary-Treasurer, North Carolina State Veterinary Examining Board, P.O. Box 9038, Asheville, N.C.

OHIO—June 6-8, 1960, Sisson Hall, College of Veterinary Medicine, Ohio State University, Columbus, Ohio. Applicants must be present at 8:00 a.m. on June 6. Dr. H. G. Geyer, Executive Secretary, Ohio Veterinary Medical Board, Ohio Departments Building, Room 720, Columbus 15, Ohio.

TEXAS—May 30-June 1, 1960, A. & M. College of Texas, College Station, Texas. The completed application must be received in the Board office not later than 30 days before the examination date. Mr. T. D. Weaver, Executive Secretary, Texas State Board of Veterinary Medical Examiners, 207 Capital National Bank Building, Austin 16, Texas.

UTAH—June 30-July 1, 1960, State Capitol Building, Department of Business Registration, Salt Lake City, Utah. Applications should be submitted to Mr. Frank E. Lees, Director of the Department of Business Regulation and Registration Division, State Capitol Building, Salt Lake City, Utah, by June 15. Registration fee is \$15.

WISCONSIN—June 27-28, 1960, Madison, Wis. Dr. A. A. Erdmann, Chief Veterinarian, State-Federal Cooperative Program, 6 West, State Capitol, Madison 2, Wis.

Deaths

Star indicates member of AVMA

Harvey Baker Hood (UP '03), 66, Hendersonville, N.C., died on Oct. 30, 1959.

Dr. Hood had served with the former BAI for many years; he had been employed as assistant state veterinarian for South Carolina from May 1921 until his retirement in July, 1953.

Following his retirement, Dr. Hood had lived in Kingstree, S.C., and later in Hendersonville, N.C.

*R. H. Jones (COL '52), 31, Helena, Mont., died Sept. 13, 1959.

Dr. Jones had been born in Sunnyside, Utah. He had conducted a general practice in Helena since his graduation from Colorado State in 1952.

*Ralph C. Swalberg (KCV '15), 74, Spanish Fork, Utah, died after a lingering illness on Dec. 20, 1959.

Dr. Swalberg had been both president of the Utah V.M.A. for two years and vice-president for a similar term. In addition, he had served as a director of the Utah State Fair Board for 12 years and was manager of the Junior Livestock Show for 11 years.

He had also been active in civic affairs and, at one time, served as mayor of Spanish Fork.

Women's Auxiliary

Highlighting . . .
Indiana—Louisiana

Indiana

INDIANAPOLIS.—The Women's Auxiliary to the Indiana V.M.A. held its sixteenth annual convention, Jan. 13-15, 1960, at the Severin Hotel in Indianapolis.

The business meeting was highlighted by reports from Mrs. Frank Booth of Elkhart, national auxiliary president; Mrs. V. K. McMahan, delegate to the Auxiliary House of Representatives; and Mrs. W. G. Magrane delegate to the International Veterinary Congress in Spain last May.

Officers elected for the coming year were: Mrs. Howard Glass, Indianapolis, president; Mrs. Harry Koeppen, Bloomington, first vice-president; Mrs. Russell Portman, Lafayette, second vice-president; Mrs. G. M. Blubaugh, Thorntown, secretary; and Mrs. W. E. Lamkin, Marion, treasurer.

A tea was held honoring Mrs. Booth and the next day, the Auxiliary held a luncheon at the Indianapolis Athletic Club. Among the entertaining elements of the convention were a program on "hand analysis," the annual banquet and dance, and a program by Purdue's glee club.

S/MRS. W. E. WELBOURN, *President*
Correspondent.

Louisiana

BATON ROUGE.—The Women's Auxiliary to the Louisiana V.M.A. held its eleventh annual meeting at Bob & Jake's Steak House in Baton Rouge, Jan. 26, 1960. The meeting was preceded by a luncheon which was pre-



Newly installed officers of the Women's Auxiliary to the Indiana V.M.A. are, left to right—Mrs. Harry Koeppen, first vice-president; Mrs. Howard Glass, president; Mrs. Russell Portman, second vice-president; Mrs. W. E. Lamkin, treasurer. Mrs. G. M. Blubaugh, secretary, is not shown.

sided over by Mrs. H. C. Melius, New Orleans, president.

During the business meeting, the publicity committee report was given by Mrs. John Morrison, Opel, in the absence of its chairman, Mrs. Philip Amy of Eunice. The membership chairman, Mrs. W. T. Oglesby, reported 96 state members, 87 national members, and seven life members.

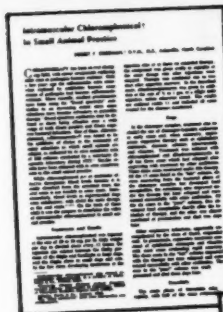
Attendees were asked to take the pamphlets, "Veterinary Medicine as a Career," for the high school libraries in their respective neighborhoods. The newspaper clipping service is being continued and members were asked to send in articles to Mrs. Amy.

The executive board contributed \$15 to the Student Loan Fund and, at the same time, initiated a committee to revise the Auxiliary's constitution and bylaws. Mrs. Willard Pounds, Metairie, was appointed its chairman.

Mrs. Melius installed the following new officers: Mrs. J. N. Thomas, Welsh, president; Mrs. Willard Pounds, Metairie, vice-president; Mrs. D. D. Magee, Kentwood, secretary-treasurer; and Mrs. W. T. Oglesby, Baton Rouge, membership chairman. Mrs. Oglesby and Mrs. Leo Pfrimmer, Franklinton, were appointed as delegates to the National Convention in Denver, Colo., next August. Mrs. A. G. Pass, of Baton Rouge, is alternate.

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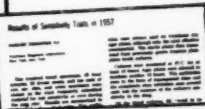
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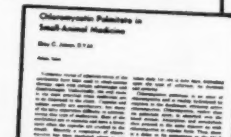
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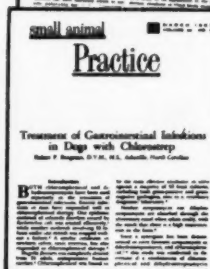
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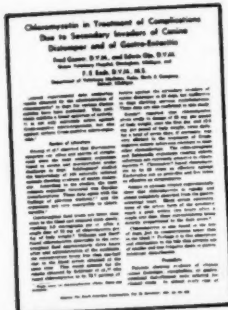
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TREATMENT OF GASTROINTESTINAL INFECTIONS IN DOGS WITH CHLOROSTREP

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CHLORAMPHENICOL AND DIHYDROSTREPTOMYCIN (CHLOROSTREP) FOR TREATMENT OF DIARRHEAS IN DOGS AND CATS

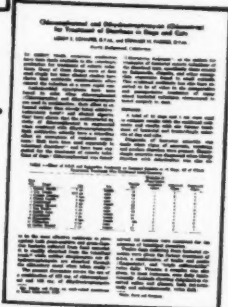
by Leroy E. Schafer, D.V.M. and Stewart H. Parker, D.V.M.

Reprint, Journal of the American Veterinary Medical Association

CHLOROMYCETIN IN TREATMENT OF COMPLICATIONS DUE TO SECONDARY INVADERS OF CANINE DISTEMPER AND OF GASTRO-ENTERITIS

by Fred Gasow, D.V.M., Edwin Oja, D.V.M.
and F. E. Ends, D.V.M., M.S.

Reprint, The North American Veterinarian



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1. Osborne, J. C. in *New Horizons in Chemotherapy. Proceedings of First Regional Conference on the Nitrofurans in Veterinary Medicine*. In press.

2. Bull, W. S.: *N. Amer. Vet.* 38: 3 (Jan.) 1957.

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Fourth Regional Conference on the Nitrofurans in Veterinary Medicine—April 1, 1960, Fairmont Hotel, San Francisco, California.

WHAT IS YOUR *Diagnosis?*

Make your diagnosis from the picture below—then turn the page ►



Fig. 1 — Lateral (left) and anteroposterior radiographs of the left hock of the Quarter horse.

History.—A 7-year-old male Quarter horse had a wire cut below the left hock a month before examination. It had not completely healed; there was still some lameness and the hock joint was badly swollen. Lateral and anteroposterior radiographs were taken.

Here Is the Diagnosis

(Continued from preceding page)

Diagnosis.—Osteomyelitis of the anterior surface of the proximal extremity of the large metatarsal bone of the horse.

Comment.—The preferred treatment would be curettage with prolonged systemic antibiotic therapy and local irrigation with antibiotics. There is sclerosis of the normal bone, surrounding the affected area with periosteal proliferation and a central sequestrum. The bone destruction extends into the medullary cavity. These findings are characteristic of osteomyelitis.

At the owner's request, the horse was euthanatized. Osteomyelitis was proved at necropsy.

This report was submitted by William D. Carlson, D.V.M., Ph.D., radiologist, College of Veterinary Medicine, Colorado State University, Fort Collins.

Our readers are invited to submit histories, radiographs, and diagnoses of interesting cases which are suitable for publication.

Quiz for Quidnuncs

1. Of what value are analeptic-sympathomimetic combinations in combating pentobarbital sodium overdosage? Page 263.
2. How valid is the rapid plate screening test in identifying leptospirosis reactors? Page 281.
3. What effects do rations and feed additive medications have on leptospirosis in cattle and swine? Page 243.
4. What is the comparative effectiveness of *Leptospira bacterin* and attenuated egg-passaged *Leptospira* vaccine? Page 254.
5. What surgical technique has been shown to be successful in correcting upward luxation of the canine scapula? Page 258.
6. What therapy proved successful in treating a dog showing signs of Addison's disease? Page 275.

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1. Jones, S. V.; Belloff, G. B., and Roberts, H. D. B.: Vet. Med. 51:413 (Sept.) 1956.

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of treated cows¹*

Fourth Regional Conference on the Nitrofurans in Veterinary Medicine—April 1, 1960, Fairmont Hotel, San Francisco, California.



Denver, Colorado Is Host to 1960 AVMA Meeting

One of the United States' fastest-growing cities, Denver, Colo., will be host to the 97th Annual Meeting of the American Veterinary Medical Association. The 1960 meeting promises to equal, if not exceed, last year's record attendance of 4,181.

Capital of Colorado and of an area known as the Rocky Mountain Empire, Denver appeals to vacationers, sight-seers, shoppers, scientists, and children filled with tales of the West and its heroes.

Since last September, the section committees have been working on a scientific program, following suggestions given by members at the section meetings at Kansas City.

Committee on Local Arrangements

Officers

General Chairman—Dr. Gail C. Gilbert, Arvada, Colo.

Vice Chairman—Dr. O. R. Adams, Fort Collins, Colo.

Secretary—Dr. Gene Bierhaus, Englewood, Colo.

Committee Chairmen

Alumni Dinners—Dr. F. Candlin, Denver, Colo.

Exhibits—Dr. R. Copeland, Denver, Colo.

Entertainment—Dr. B. Frank, Sterling, Colo.; Dr.

V. Stauffer, Arvada, Colo.

Garages—Dr. F. Judish, Denver, Colo.

Golf—Dr. D. Albrecht, Denver, Colo.; Dr. B. S. Burkhardt, Denver, Colo.

Hotels—Dr. R. Tolley, Englewood, Colo.

Meeting Rooms and Equipment—Dr. H. Schaulis, Denver, Colo.

Publicity—Mr. Claude Ramsey, Denver, Colo.

Reception—Gen. Wayne O. Kester, Denver, Colo.

Registration—Dr. Don LyVere, Denver, Colo.

Television—Dr. L. Phillips, Lakewood, Colo.

Women's Activities—Dr. L. Ramsay, Englewood, Colo.

Hotels and Housing Bureau

The Denver Hilton, one of the nation's newest hotels, has been selected as the headquarters hotel for the meeting. The pre-convention sessions of the Executive Board and House of Delegates will be held there.

Eighteen hotels have been selected to house convention registrants. All rooms marked with an asterisk on the reservation form will be air-conditioned. Most hotels are within walking distance of the Denver Municipal Auditorium where the program sessions will be held.

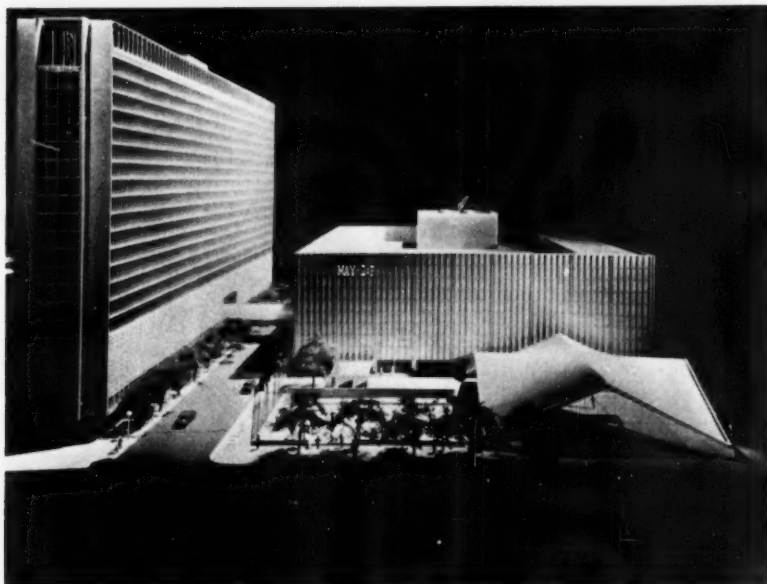
Hotel information, rates, and reservation form will be found on adv. pages 53 and 54 of this issue. A location map for hotels and some other centrally located points of interest will be found on adv. page 52.

Against the spectacular backdrop of the Rocky Mountains, the Denver skyline provides a gleaming panorama of man-made beauty. Prominent buildings in picture include Colorado's gold-domed State Capitol building (far left), Denver Civic Center (left center), the new Denver Hilton Hotel and Mile High Center (center), the new Brown Palace West Hotel, the Denver Club, and the 28-story First National Bank building, Denver's highest (far right).

ents at no extra charge. If more than one room is required to accommodate children, the hotels will charge only the single rate for each room.

Motels

A number of excellent motels are located in the Denver area and reservations may be



An architect's sketch of the completed Denver Hilton (left) shows the relationship between the AVMA headquarters hotel and one of Denver's many fine department stores. The city's shopping district, just a short distance from the hotels, awaits your approval as an outstanding fashion center.

The Denver Convention and Visitors Bureau will handle reservations and is now ready to function. Early reservations are advised.

"Family Plan" Available

The 18 hotels selected for convention housing offer a "family plan" whereby children under 14 years of age will be accommodated in the same room with their par-

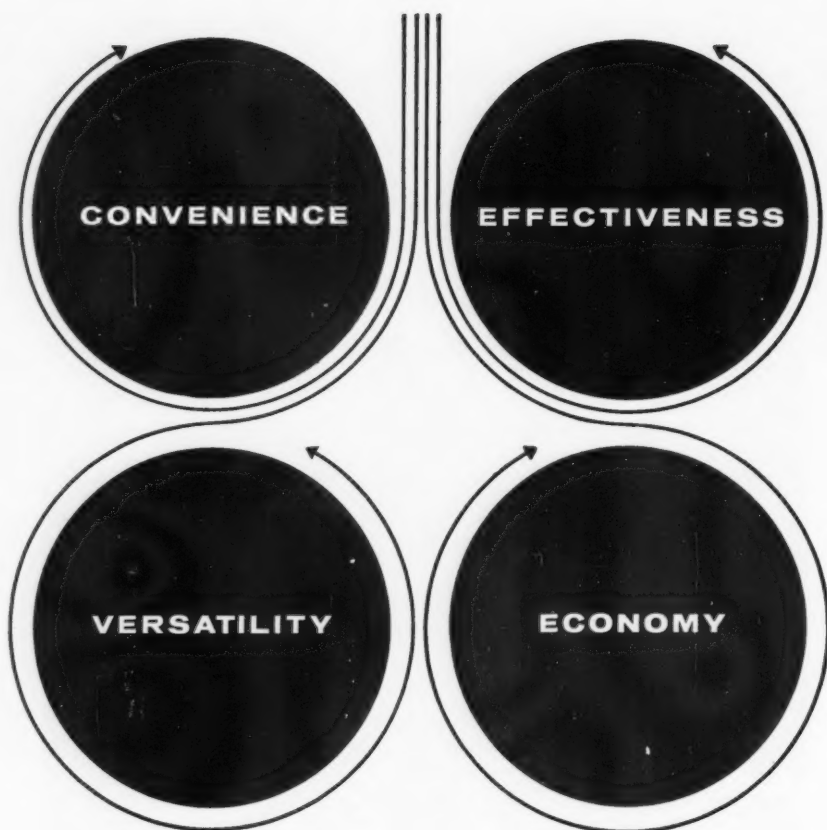
made through the Denver Convention and Visitors Bureau, 325 W. Colfax Ave., Denver 2, Colo.

APPLICATIONS

Applicants Not Members of Constituent Associations

In accordance with paragraph (c) of Section 1, Article I, of the Bylaws, the names of applicants who are not members of constituent associations shall be published in the JOURNAL. Written comments re-

(Continued on adv. p. 45)



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(Applications—continued)

ceived by the Executive Secretary from any active member regarding the application as published, will be furnished to the Judicial Council for its consideration prior to acceptance of the application.

VAKILZADEH, JAVAD

North Carolina Sanatorium System
Chapel Hill, Nor. Car.
D.V.M., Univ. of Teh., 1952
Vouchers: L. L. Vine and R. F. Chambless

FRESHOUR, DONALD F.

168th Med. Det. VFI
APO 742, New York, N. Y.
D.V.M., M. S. U., 1936
Vouchers: Curtis W. Betzold and Charles B. Frank

LEVINGSTON, SAMUEL W.

51 Med. Det. (VAH)
APO 108, New York, N. Y.
D.V.M., Texas A. & M. College, 1955
Vouchers: Curtis W. Betzold and Charles B. Frank

WOLFORD, STEPHEN T.

U. S. Army Medical Service Group,
Fort Buckner, APO 331
San Francisco, Calif.
D.V.M., O. S. U., 1956
Vouchers: Millard L. Tierce and H. J. Magens

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During 1960, cats will solidify their position as America's most popular pet. An estimated 28 million cats, compared with 26 million dogs, are adopted by nearly one fourth of all American families.

Although cats are more frequently found in suburban and rural areas, the fancier breeds, such as Siamese and Burmese, are favored in the cities. Besides being clean, quiet, and easily housebroken, a good cat can destroy more than a dozen mice a night.—Release, Quaker Oats Co., Chicago.



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History of the AVMA

The affairs of the USVMA reached a low ebb in 1882, when at the Boston meeting it was rather euphemistically reported: "Fully one-third of the members were present [about two dozen], two new members were elected, and the regular committees had no reports to offer . . . E. F. Thayer presented some remarks on an osseous nasal polypus which he successfully removed in 1867 [!]. . . The discussion following on the use of the wire ecraseur [introduced by Jennings in the 1850s] was of much interest . . . The meeting then adjourned to dinner, after which the subjects of splenic fever, Texas fever and quitters were discussed."

As usual, the annual meeting was held at the American Veterinary College and the officers of the previous year were re-elected. Whether Liautard's earlier charge that the main fault of the Association was in its officers was directed at Wm. Bryden in particular is a moot point, but it is obvious that little transpired during his tenure as president. However, bowing to custom, his re-election was reassured.

Perhaps as a rebuke to the regular committees, which had nothing to report, Dr. Liautard, as chairman of the Committee on Diseases, states: "Being unexpectedly called to Europe this summer . . . I was almost tempted to give up the work and follow the example of our friendly predecessors and only report 'progress,' but at the same time thought that in so doing I would do injustice to our friends and to the Association." Accordingly, he had

distributed 100 copies of a questionnaire to members in 29 states; 30 replies from 15 states were returned. In order of frequency of mention, the most common diseases were: equine influenza, glanders and farcy, anthrax, Texas fever, hog cholera, chicken cholera and cerebro-spinal meningitis.

As reported by Secretary Michener: "Dr. Liautard then introduced the subject of inoculation for anthrax. He also presented to the society the different instruments chiefly used in inoculating cattle and sheep. Anthrax blood and virus were shown in hermetically sealed tubes [*virus* was then used as *infective agent*]. Prof. Liautard suggested an appropriation of society funds for experimental purposes. It was afterward resolved that a committee of three be appointed to make experiments on the value of inoculation by the method of Pasteur, and \$150 was appropriated for the purchase of some sheep and cows for this purpose. Dr. Liautard was appointed chairman of the committee, with power to select the other two members."

Although a pre-convention notice had stated: "There is no doubt that besides the reports of the various committees, several interesting papers will be presented," the Secretary's report of the meeting states: "There were no reports [by three of four committees], and . . . there were no regular papers presented." Drs. Stickney and W. B. E. Miller presented two cases of osteomalacia, but "other reports of cases were denied the Association owing to the lateness of the hour. The society adjourned to a banquet at Delmonico's, where the evening was passed in the most social and pleasant manner."



WILLIAMSON BRYDEN, V.S., 11th President of the USVMA, was born in Scotland and came to this country when a boy. He was graduated from the Montreal Veterinary College in 1871 and served as a member of the board of examiners of that school for a number of years. He was a charter member and president of the Massachusetts VMA, and served as president of the USVMA 1881-1883. For many years he was an inspector of cattle at the port of Boston. Upon his death June 28, 1895, he was characterized as "a good friend and ever-ready adviser . . . of marked intelligence, and an able student and practitioner."



Dr. C. P. Lyman, USVMA president, 1877-1879

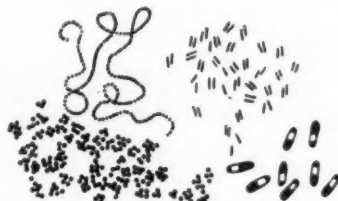
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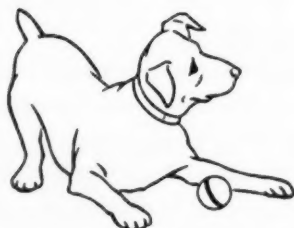
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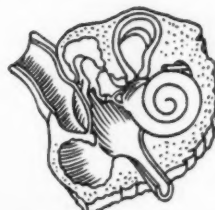
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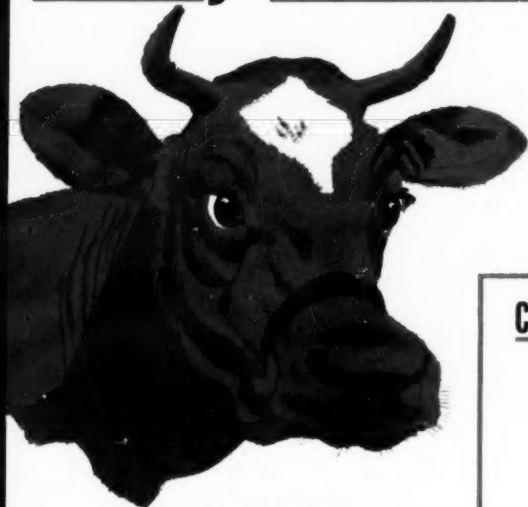
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Dosage may be adjusted according to condition treated and effect desired. For long-term therapy, dosage may be decreased gradually to one tablet daily or as required.

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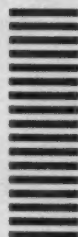
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HOTEL INFORMATION—DENVER, COLORADO, CONVENTION

Ninety-Seventh Annual AVMA Meeting, Aug. 14-18, 1960

All requests for hotel accommodations will be handled by a Housing Bureau in cooperation with the Denver Convention and Visitors Bureau. The Bureau will clear all requests and confirm reservations.

Hotel and Rate Schedule

(See Location, by Number, on Map of Downtown Area)

Map No.	Hotel	Single (1 person)	Double bed (2 persons)	Twin bed (2 persons)	Suites	Sets-2 rooms connecting bath (2-3-4 persons)
1	Adams*	\$5.50-7.50	\$ 7.50-9.00	\$ 8.50-10.50	\$13.50-15.00	\$ 9.50-16.00 (1 Room, 2dbl. beds)
2	Albany†	6.50-9.50	10.00-12.00	12.50-14.00	30.00	- - - - -
3	Ambassador	5.50-6.00	7.00-7.50	9.00	- - - - -	- - - - -
4	Argonaut*†	6.50-9.50	8.50-11.00	9.50-12.50	- - - - -	13.50-17.50
5	Auditorium*	5.00	6.50	7.00	- - - - -	8.00-12.00
6	Broadway Plaza†	8.00-10.00	10.00-12.00	12.00-14.50	18.00-28.00	- - - - -
7	Brown Palace†	9.00-15.00	13.00-17.00	14.00-19.00	22.00-70.00	18.00-22.00
8	Colorado*	4.50-6.00	6.00-10.00	8.00-12.00	- - - - -	14.00-20.00
9	Cory	5.00-7.00	6.00-9.00	6.50-9.00	- - - - -	- - - - -
10	Cosmopolitan*	8.50-11.00	12.00-18.00	14.00-20.00	22.00-60.00	- - - - -
11	Hillview	9.00-11.50	10.00-12.00	12.00-13.50	16.00-18.00	- - - - -
12	Hilton*†	HEADQUARTERS HOTEL — Reserved exclusively for official convention use.				
13	Kenmark (not a/c)	4.50-6.50	6.00-7.00	7.50-8.00	- - - - -	6.00-12.00
14	Mayflower*	7.50-14.50	8.50-16.50	12.50-18.50	- - - - -	- - - - -
15	Olin*	5.00-7.00	9.00-11.00	10.00-12.00	- - - - -	11.00-16.00
16	Oxford	5.00-10.00	6.50-10.00	8.50-11.00	13.00-16.00	- - - - -
17	Sears	5.00-6.00	6.50	7.50	- - - - -	14.00 (For 2-3-4 persons)
18	Shirley Savoy*	7.00-9.00	9.50-11.50	11.00-13.00	25.00	15.00-19.00

† 100 per cent air-conditioned; in other hotels listed, majority of rooms air-conditioned.

***FAMILY PLAN**—The above hotels offer a "family plan" whereby children under 12 years of age will be accommodated in the same room with their parents at no extra charge. If more than one room is required to accommodate children, the hotel will charge only the single rate for each room.

MOTELS—Reservations for motels in the Denver area may be made through the Denver Convention and Visitors Bureau, 225 West Colfax, Denver 2, Colo.

PLEASE USE APPLICATION ON REVERSE SIDE FOR HOTEL ACCOMMODATIONS

Application for Hotel Accommodations

1960 AVMA Convention — Denver, Colorado

The Convention and Visitors Bureau will make every effort to place you according to your expressed wishes or, if the accommodations of your choice are not available, the Housing Bureau will select one that is nearest to the preferred rate and location.

Please give us the complete information requested below. At least four choices of hotels, or more if you desire, are necessary. Arrange for double occupancy of rooms wherever possible; only a limited number of single rooms is available.

Date

Hotel First Choice

Hotel Second Choice

Hotel Third Choice

Hotel Fourth Choice

☐ Room with bath for one person. Rate per room desired \$..... to \$.....

☐ Room with bath for persons. Rate per room desired \$..... to \$.....

☐ double bed ☐ twin beds

☐ Two rooms with connecting bath for persons:

Rate per set desired \$..... to \$.....

☐ Suite with bedroom(s) with bath for persons:

Rate per suite desired \$..... to \$.....

☐ Check here if you desire accommodations on the FAMILY PLAN.

Arrival date, hour A.M. P.M.

Departure date

If reservations cannot be made in one of the hotels indicated shall we place you elsewhere? ☐ Yes ☐ No

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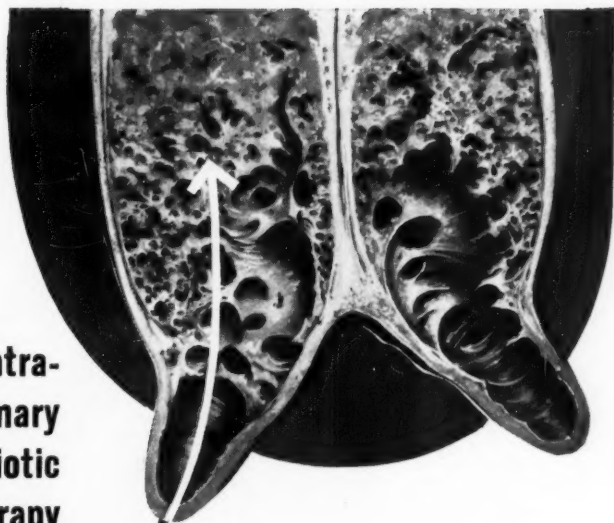
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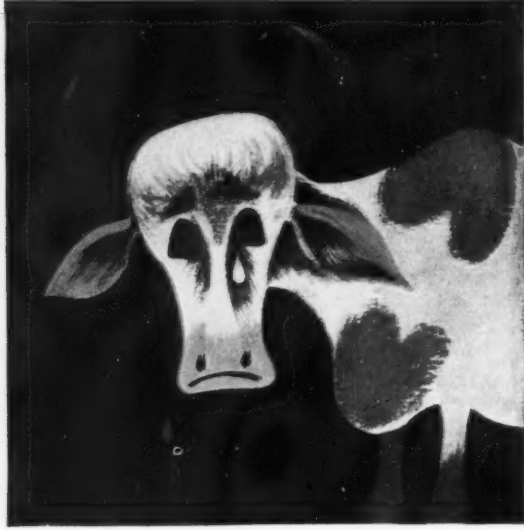
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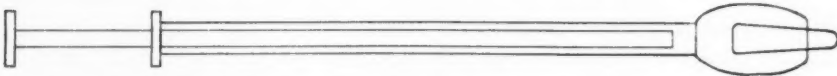
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1. Vigue, R. F., et al.: J. Am. Vet. M. Ass. 134:308 (April 1) 1959.

2. Vigue, R. F.: Personal communication.

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COMING MEETINGS

Notices of coming meetings must be received
30 days before date of publication.

New Jersey Veterinary Medical Association. Seventy-sixth annual meeting. Hotel Claridge, Atlantic City, March 30-31, 1960. John R. McCoy, 236 Highway 18, East Brunswick, N. J., secretary.

Alabama Veterinary Medical Association. Annual meeting. Battle House, Mobile, April 3-5, 1960. M. K. Heath, School Veterinary Medicine, Auburn University, Auburn, Ala., secretary.

National Institute of Animal Agriculture. Tenth annual meeting. Purdue University, Lafayette, Ind., April 3-5, 1960. Claude Harper, Department of Animal Science, Purdue University, Lafayette, Ind., treasurer.

Florida, University of. Third annual conference for veterinarians. University of Florida, Health Center Auditorium, April 30-May 1, 1960. W. R. Pritchard, Head, Department of Veterinary Science, University of Florida, Gainesville.

Kansas State University. Twenty-second annual conference. School of Veterinary Medicine, Kansas State University, Manhattan, May 19-21, 1960. Donald C. Kelley, chairman.

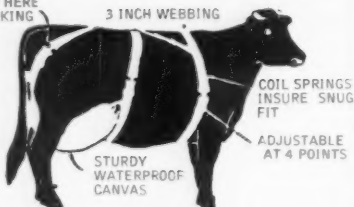
Texas, A. & M. College of. Thirteenth annual conference for veterinarians. School of Veterinary Medicine, A. & M. College of Texas, College Station, June 2-3, 1960. R. D. Turk, conference chairman.

(Continued on adv. p. 64)

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Mississippi State Veterinary Medical Association, Inc. Annual meeting. King Edward Hotel, Jackson, June 19-21, 1960. Joseph W. Branson, P. O. Box 4223, Fondren Sta., Jackson, Miss., secretary-treasurer.

Maryland State Veterinary Medical Association. Summer meeting. George Washington Hotel, Ocean City, June 23-24, 1960. Harry L. Schultz, Jr., 9011 Harford Rd., Baltimore 14, Md., secretary.

California Veterinary Medical Association. Seventy-second annual meeting. Jack Tar Hotel, San Francisco, Calif., June 26-29, 1960. Mr. Ken Humphreys, 3004 16th St., San Francisco 3, Calif., executive secretary.

Virginia Veterinary Medical Association. Summer meeting. Shoreham Hotel, Washington, D.C., July 17-19, 1960. G. B. Estes, State Office Building, Richmond, Va., secretary-treasurer.

Auburn University. Fifty-third annual conference for veterinarians. School of Veterinary Medicine, Auburn University, July 24-27, 1960. J. E. Greene, dean.

American Veterinary Medical Association. Ninety-seventh annual meeting. Denver-Hilton Hotel, Denver, Colo., Aug. 15-18, 1960. H. E. Kingman, Jr., 600 S. Michigan Ave., Chicago 5, Ill., executive secretary.

Foreign Meetings

International Association of Veterinary Food Hygienists. Second Symposium. Basel, Switzerland, May 15-21, 1960. Dr. A. Clarenburg, 1, Sterrenbos, Utrecht, The Netherlands, president.

International Congress of Physio-Pathology of Animal Reproduction and Artificial Insemination. Amsterdam, Netherlands, June 13-17, 1960. Dr. J. Edwards, Milk Marketing Board, Thames, Surrey, England.

First International Congress of Endocrinology. Technical University of Denmark, Copenhagen, July 18-23, 1960. Dr. Christian Hamburger, Statens Seruminstitut, Copenhagen S, Denmark, chairman of the executive committee.

Note: Regularly scheduled meetings will be published in the first-of-the-month issues only.

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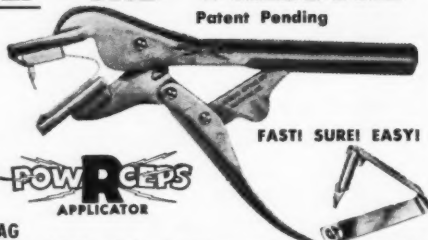
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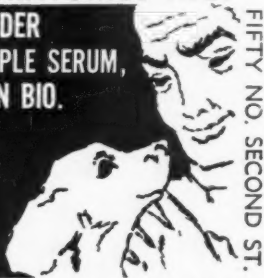
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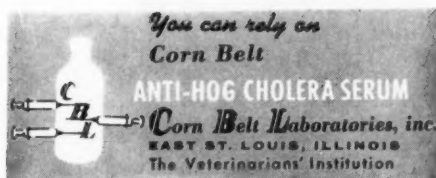
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